"CLINICAL PROFILE, & OUTCOME OF TOXIN INDUCED ACUTE KIDNEY INJURY"

Dissertation submitted in partial fulfillment of the requirements for the degree of

D.M. (NEPHROLOGY)

BRANCH- III

DEPARTMENT OF NEPHROLOGY

MADRAS MEDICAL COLLEGE

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THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

AUGUST 2014

DECLARATION

I, Dr. **JEGAN.A**, solemnly declare that the dissertation titled "**CLINICAL PROFILE, & OUTCOME OF TOXIN INDUCED ACUTE KIDNEY INJURY"** is the bonafide work done by me at Department of Nephrology, Madras Medical College under the expert guidance and supervision of Dr. N.GOPALAKRISHNAN M.D., D.M., FRCP, Professor of Nephrology, Madras Medical College. The dissertation is submitted to the Tamilnadu Dr.M.G.R Medical University towards partial fulfillment of requirement for the award of D.M. Degree (Branch III) in Nephrology.

Place: Chennai **Dr. JEGAN.A**

Date:

CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL PROFILE, & OUTCOME OF TOXIN INDUCED ACUTE KIDNEY INJURY"** is a bonafide work done **Dr.JEGAN.A**, Department of Nephrology, Madras Medical College, in partial fulfillment of the University rules and regulations for award of D.M., Nephrology under my guidance and supervision during the academic year 2011 – 14**.**

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CONTENTS

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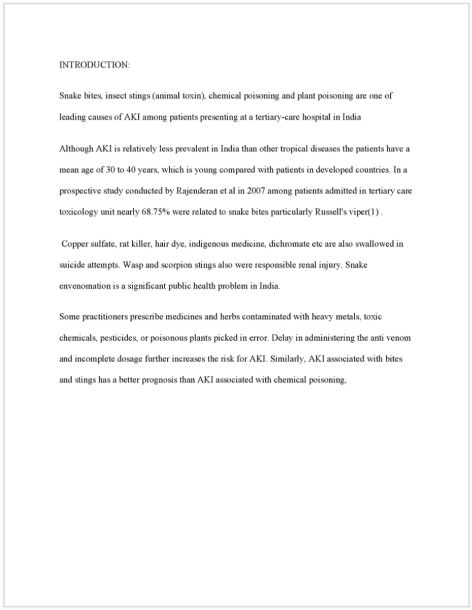


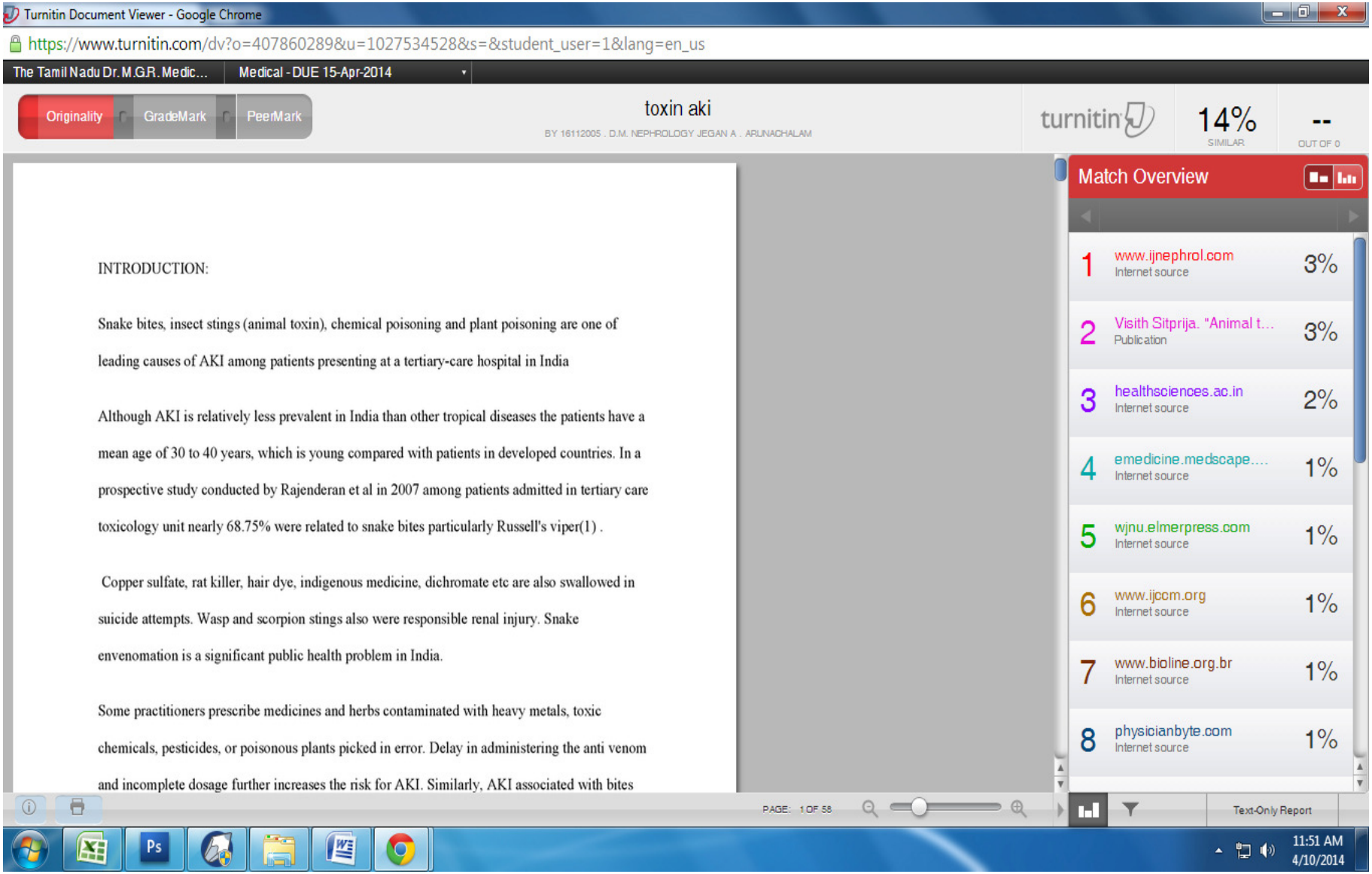
INTRODUCTION:

Snake bites, insect stings (animal toxin), chemical poisoning and plant poisoning are one of leading causes of AKI among patients presenting at a tertiary-care hospital in India

Although AKI is relatively less prevalent in India than other tropical diseases the patients have a mean age of 30 to 40 years, which is young compared with patients in developed countries. In a prospective study conducted by Rajenderan et al in 2007 among patients admitted in tertiary care toxicology unit nearly 68.75% were related to snake bites particularly Russell's viper(1) .

Copper sulfate, rat killer, hair dye, indigenous medicine, dichromate etc are also swallowed in suicide attempts. Wasp and scorpion stings also were responsible renal injury. Snake envenomation is a significant public health problem in India.

Some practitioners prescribe medicines and herbs contaminated with heavy metals, toxic chemicals, pesticides, or poisonous plants picked in error. Delay in administering the anti venom and incomplete dosage further increases the risk for AKI. Similarly, AKI associated with bites and stings has a better prognosis than AKI associated with chemical poisoning.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ABSTRACT

AIM OF STUDY

To study the clinical profile and outcome in toxin induced acute kidney injury. To find out incidence of AKI among poison cases.

MATERIALS AND METHODS

In this study we had selected all cases who had developed renal failure after toxin ingestion or animal bite (snake, wasp sting and scorpion). In this prospective study cases admitted to the Poison Control, Training and Research Centre of Government General Hospital, Madras Medical College were monitored and evaluated for development of AKI.

RESULTS AND OUTCOME

Total number of cases during the study period was 4125. Total number of death during the period was 264(6%). Total case presented with renal failure is 178(4.3%).Total number of cases with dialysis requirement is 130(83%).Total number of cases not requiring dialysis was 48 (17%).Total number of males is 115(64%)Total number of females 63(36%) .Total number of death in AKI IS 45(17%).most common cause of death is paraquat33%

CONCLUSION

Incidence of AKI in our study population was 178 (4.3%) of which dialysis requirement was seen in 130 (83%). Snake bite was the leading cause of toxin induced AKI (46%). Risk factors for the development of AKI in this population include cellulitis, regional lymphadenopathy, presence of fang marks, while predictors of poor outcome were hypotension, DIC and rhabdomyolysis. Early adequate administration of snake anti-venom had reduced the severity of AKI. Type of snake bite particularly pit viper bite had very poor outcome. Paraquat and wasp had very high incidence and mortality among the study group.

INTRODUCTION:

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Some practitioners prescribe medicines and herbs contaminated with heavy metals, toxic chemicals, pesticides, or poisonous plants picked in error. Delay in administering the anti venom and incomplete dosage further increases the risk for AKI(2). Similarly, AKI associated with bites and stings has a better prognosis than AKI associated with chemical poisoning,

AIM OF STUDY

- To study the clinical profile and outcome in toxin induced acute kidney injury
- To find out incidence of AKI among poison cases.
- To do histopathological study in needed cases and its correlation with the outcome.
- To study about correlation of early decontamination and severity of renal failure.
- To follow up the patients and to find out the incidence of CKD in this group.

REVIEW OF LITERATURE

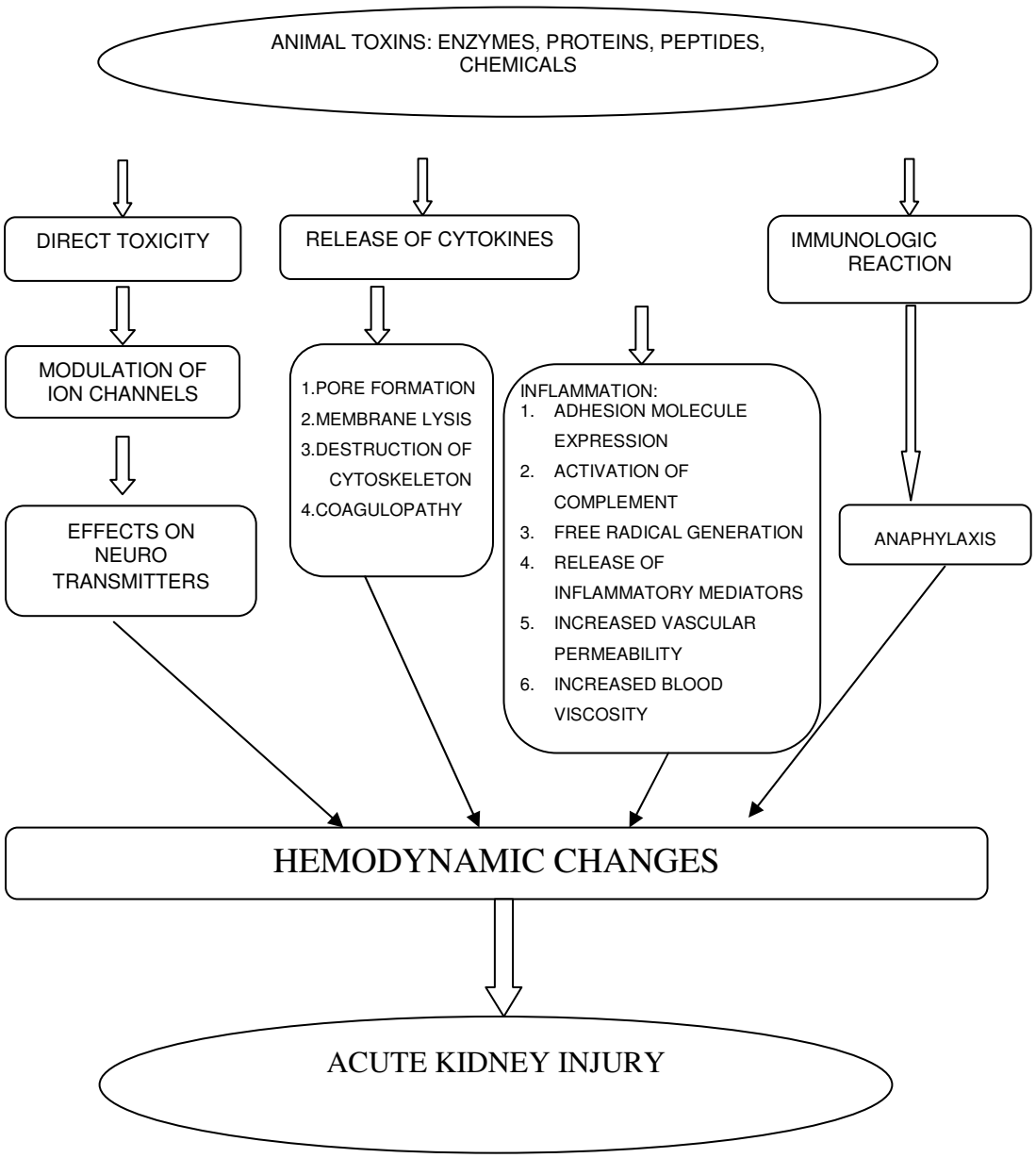
SNAKE BITE

Snake bite is one of the common preventable public health hazard in tropical and subtropical countries with dense vegetation and vast tracts of agricultural land. India have the highest estimated snake bite mortality in the world (WHO) placing the number in between 15,000 to 30,000 per annum. Some experts think that it could be between 10,000 to 50000 per annum(2). Discrepancy in data is because of our very poor system of entry, reporting of morbidity and mortality and retrieval of documents..The most common venomous snakes in India are Indian or spectacled cobra, Common krait, Russell's viper, Saw-scaled viper, Hump-nosed pit viper White lipped pit viper and sea snake(1,12,13).

Pathophysiology: More than hundred different types of peptides or proteins, lipids, amines, carbohydrates etc. have been isolated from snake venoms, not all of which are toxic to humans. Endothelial disruption and activation or inhibition of coagulation proteins or platelets, caused by phospholipases, serine proteases, disintegrins, metalloproteinases and C-type lectins leads to coagulopathy. (4,12).

Hemorrhagins causes bleeding by directly injuring the vascular endothelium. Hyaluronidase, Hydrolases and peptide cytotoxins contribute to the local tissue necrosis. Myotoxic phospholipase A2seen in sea snakes is responsible for rhabdomyolysis which can later lead to acute renal failure. Permeability factors

(bradykinin like peptide(4), endothelin like toxin, VEGF and natriuretic peptides)that increase extravastion of plasma, and effects on cardiac and vascular smooth muscle can lead to hypotension without any bleeding manifestations. Neurotoxins impair transmission at NMJ by acting either pre synaptically or post synaptically.



AKI is common after hemotoxic or myotoxic snakes bites.(2) The incidence of AKI varies from5% to 29% depending on the species and the severity of envenomation.(4,5) The onset of renal failure is from a few hours to as late as 96 h after the bite and the duration ranges from 2 to 3 weeks. Prolonged AKI with oligoanuria after snake bite is an indication for renal biopsy which usually reveals cortical necrosis or acute tubular necrosis associated with interstitial nephritis or extracapillary glomerulonephritis(9,10). Clinical recovery from snake-bite-induced AKI is usually complete, unless cortical necrosis is present. Mortality in snake-bite-related AKI ranges from 1% to 20%.(3,11,13). Extremes of age ,female gender, hemodynamic instability, DIC, thrombocytopenia, delayed anti-venom treatment, native medication, applying tourniquet,and presence of corticalnecrosis are associated with poor prognosis.(13) Although monospecific antivenom administration is the strategy of choice since species identification is difficult in most of the times polyvalent anti venom specific to India is being commonly used.If AKI is already established, early and frequent peritoneal or hemodialysis is lifesaving. Muscular symptoms associated with sea snake are improved by hemodialysis. Forced alkaline diuresis,if performed early can prevent AKI in patients with myoglobinuria or hemoglobinuria.(21,23) In established AKI, administration of sodium bicarbonate or mannitol will be life threatening**.**

AKI is common after bites from hemotoxic or myotoxic snakes.the mechanism involved are Direct toxicity, Hemodynamic changes,Intravascular hemolysis,Disseminated intravascular coagulation, Immunologic reaction and Rhabdomyolysis

PATHOLOGY

The most common renal pathology is ATN .The other possible renal pathology according to various literature are given below

- Acute tubular necrosis
- Acute cortical necrosis
- Interstitial nephritis
- Thrombotic microangiopathy
- Mesangiolysis
- Vasculitis
- Glomerulonephritis
- Renal infarction

Acute cortical necrosis is the most dreaded histopathological finding as it confers very poor renal survival.4. Snake bite is the second common cause of acute cortical necrosis in India following obstetric causes.(28) Primary glomerular involvement is uncommon. Mesangiolysis is an early and most consistent abnormality associated with snake bite envenomation(23).Mild focal and segmental mesangial proliferation is common. Severe form of crescentic or diffuse proliferative glomerulonephritis is very rare

ANTI SNAKE VENOM IN INDIA

The ASV is prepared serum institute of India from hyper immune horses against the venoms of the most commonly encountered poisonous snakes in India. Each ml. contains

Cobra	0.6 mg
Common-Krait	0.45 mg
Russell's Viper	0.6 mg
Saw scaled Viper	0.45 mg

HYMENOPTRA

This order is sub divided into aspedia, vespidea, formecedae. Wasp, hornet and yellow jacket are members of the vespid subgroup(14). Stings are usually associated with only local reactions, rarely anaphylaxis, hemolysis, rhabdomyolysis, thrombocytopenia, elevated hepatic serum enzymes, acute renal failure and *MODS. Onset of symptoms* will be within 24 hours or be delayed.([16] Initially they may have mild symptoms and develop severe acute clinical manifestation later. Clinicians should be aware of possible complications of wasp stings in order to provide early detection and proper management.Cardiac involvement is characterized both by a ischemia and arrhythmia. Respiratory manifestations included laryngeal edema, pulmonary edema and pneumonia. Hematologic involvement included intravascular hemolysis, leukocytosis, thrombocytopenia and coagulopathy.[4,21-23] Direct toxicity

to muscles is considered the main cause of rhabdomyolysis and subsequent myoglobinuria may be an important cause of acute kidney injury. A CPK level >5,000 U/L is associated with AKI. Acute kidney injury is common and most serious complication. Renal damage may be caused by Direct toxin nephrotoxicity,Hypotension ,Hemoglobinemia,Myoglobinemia. Renal pathology has mainly shown acute tubular necrosis and acute interstitial nephritis.(14,15) Renal recovery in acute interstitial nephritis was much longer than in acute tubular necrosis (66.5 versus 35.6 days) and may result in interstitial fibrosis and irreversible renal failure(15) Renal biopsy should be done early since AIN will respond dramatically to early steroid therapy. Renal tubular acidosis, a rare complication, has also been reported(34,35,36).

SCORPION STING

Scorpions live in warm dry regions in India. Reports from Bellary, Rayalaseema, rural Maharashtra, Pondicherry, madras, and vellore have documented such fatalities in children and adults . Scorpion venoms are complex mixtures of amino acids, lipids, mucopolysaccharides, hyaluronidase, 5-hydroxytryptamine,histamine phospholipase, and neurotoxic peptides(17,18,19). Scorpion venoms stimulate autonomic nervous system by opening sodium channels, which leads to hemodynamic alterations. AKI after scorpion sting has been described in association with DIC, hemolysis, bleeding in various organs, and cardiovascular complications.

PATHOLOGY

The renal pathological changes observed include renal tubular cell degeneration, glomerular basement membrane damage and interstitial hemorrhage.(19) Hemolytic uremic syndrome has also been reported after scorpion sting. In severe cases, early treatment with antivenom can prevent renal and cardiovascular complications.

INGESTION OF RAW CARP BILE

The raw bile of carp from the family Cyprinidae contains dependent nephrotoxic molecules, including cyprinol(38). The amount of bile ingested varies from 15 ml to 30 ml. Symptoms begin as abdominal pain, vomiting and diarrhea 10 min to 12 h after ingestion; jaundice and AKI follow in 62% and 54% of patients, respectively(39,40). AKI develops within 2–48 h of ingestion and for 2–3 weeks. Renal pathology shows tubular necrosis. The toxic ingredient is 5 α-cyprinol sulphate, a 27-carbon bile salt. Renal and liver biopsies will show acute tubular necrosis and hepatocellular injury (42). It has been postulated that by inhibiting the cytochrome c oxidase, toxin promots calcium influx, and inducing lysosome membrane instability.() Acute renal failure accounts for more than 80% of the deaths (42). Other manifestations include cardiac complications, hemolysis, convulsion and coma (42). Treatment is mainly supportive and Renal replacement therapy.

PLANT TOXINS

There are thousands of plants around the world, which are lethal to the humans.In india plant toxins that are of renal significance are *Cleistanthus collinus*, Thevetia peruviana (Yellow Oleander) and abrus precatorius.

THEVETIA PERUVIANA (YELLOW OLEANDER)

Cardiac glycosides are found in a diverse group of plants including Yellow Oleander **.**The more than 200 naturally occurring cardiac glycosides have been identified. Toxin will bind to a site on the cell membrane, producing reversible inhibition of the Na^+-K^+ ATPase. This increases intracellular Na^+ and decreases intracellular K^+. Elevated intracellular Na^+ concentrations produce increased intracellular calcium concentrations via an Na^+ -(Ca^{++})-exchanger. In response to the increased intracellular Ca^{++} the sarcoplasmic reticulum releases more calcium intracellularly, resulting in depolarization of the cell.(43)As a result of this enhanced cardiac contractions, which are delayed after depolarization occurs. Cardiac glycosides primarily affect cardiovascular, neurologic, and gastrointestinal systems.(43)

CLEISTANTHUS COLLINUS POISONING

Distal renal tubular acidosis with hyperchloremic normal anion gap metabolic acidosis; alkaline urine; and hypokalemia with kaliuresisis the most common renal syndrome.[12] A detailed study of renal tubular function revealed that the distal tubules are the most susceptible although proximal tubular injury can

occur, and in more severe forms, global tubular dysfunction with diminished glomerular filtration rate (GFR) may occur. Metabolic acidosis with defective urinary acidification often persists even at discharge. Renal failure is oliguric in most instances and its etiology is probably multifactorial: direct toxin effect secondary to hypotension and, possibly, secondary to hypokalemic rhabdomyolysis(12). Hyponatremia has also been documented. Hypomagnesemia have an additional role in hypokalemia refractory to treatment.

PARAQUAT POISONING

Paraquat (bipyridilium herbicide) is inactivated by adsorption to clay in the soil(44). The toxicity is through redox cycling, leading to generation of superoxide anions which react to form hydrogen peroxide and subsequently the highly reactive hydroxyl radical(44,45,46). This is thought to be responsible for lipid peroxidation and cell death. A second contributing factor to toxicity is the depletion of (NADPH), as both hydrogen peroxide detoxification as well as paraquat redox cycling via glutathione is NADPH dependent(44,45,51). The clinical course of paraquat poisoning depends upon the amount ingested and the time lag between ingestion to hospitalization(early decontamination, extracorporeal therapy, cyclophosphamide pulse) .

Mild poisoning occurs with ingestion of <20 mg of paraquat per kg body weight (<1.5 g). In moderate poisoning, ingestion is around 20–40 mg/kg (1.5–3 g). Severe poisoning occurs with ingestion of 40–80 mg/kg (3–6 g) .Fulminant poisoning is seen after ingestion of more than 80 mg/kg of paraquat (>6 g). After

ingestion, maximum concentration is found in the lungs and the concentration peaks in 5 – 7 hours .

Hemoperfusion (HP) (48,49,51)using activated charcoal if initiated within 4 hours of paraquat ingestion is thus effective and there is no indication of repeated HP . Plasma levels peak early in the course, then decrease rapidly during the first 10 hours after ingestion because of paraquat distribution to tissues . The clinical course of AKI is like oliguric acute tubular necrosis.

The precipitating factors of renal failure are multifactorial including delayed referal, significant GI fluid losses, circulatory failure, multi-organ failure and septicemia. Since > 90% is eliminated by the kidneys as the parent compound, the mechanism of AKI is assumed to be related to redox cycling and oxygen toxicity 6 but this has not been proven. There is no proven antidote for paraquat poisoning. Because of free radical injury is in lungs, some groups advocate routine use of antioxidants (Vitamin C 4000 mg/day and Vitamin E 250 mg/day) even though there is no data to support.

Pulse therapy using cyclophosphamide and steriods has been shown to be effective in preventing pulmonary fibrosis(51,52). As oxygen potentiates lung injury, supplemental oxygen should not be given until FIO2 falls below 50 . Recently there has been some interest in using NO to treat paraquat-poisoning . Paraquat specific IgG antibodies and their Fab fragments are effective though the "window of opportunity" is very short . Lung transplantation has been performed in a few patients. The mortality of paraquat poisoning remains high. MODS with

circulatory collapse are associated with 100% mortality in acute stage while late onset pulmonary fibrosis with respiratory failure also remains an important cause of mortality (49). The survival rate in is around 40 to 50%.

HAIR DYE POISONING

The major component of hair dye is (para-phenylenediamine , an aromatic amine).(53,54,55) The extent of renal involvement varies from mild transient proteinuria to oliguric acute kidney injury. AKI commonly develops a few days after exposure. The mechanisms of kidney injury following hair dye poisoning are due to direct toxic effect on kidney It cause rhabdomyolysis with deposition of myoglobin cast within the renal tubules and hemolysis with hemoglobinuria causing ATN(55,56,57).The hypovolemia and hemodynamic instabity (due to direct myocardial deppresant activity and cardiac muscle rhabdomyolysis) also predisposes to AKI. The development of AKI in PPD intoxication varies from 47.3% to 100%

COPPER SULPHATE POISONING

The incidence of copper sulphate poisoning with the availability of the poison. Its more common in the urban areas with large number of small scale industries like northern parts of Chennai, Thirupur and Thiruvallur districts in Tamilnadu. The mortality rates vary from 14-18.8%. In a study from Aligarh in 1970's, it was the commonest mode of poisonings at that center accounting to 118 cases over four and a half years. However, the incidence of copper sulphate poisoning is declining in now a days in all parts of India. It is proposed that free

reduced copper binds to sulfhydryl groups and inactivates enzymes such as G6PD and glutathione reductase.

In addition copper may interact with oxygen species like superoxide anions and hydrogen peroxide and catalyze the production of toxic hydroxyl radicals. Lethal dose is about 10-20 g. Renal complications are usually seen on the third or the fourth day. In a report of AKI manifestations following copper sulphate poisoning, histology of kidney revealed features of acute tubular necrosis in seven out of eight kidney biopsies and tubules contained hemoglobin casts. A single case of interstitial granuloma was also reported.

RODENTICIDE POISONING

Anticoagulant rodenticides are poisons used to kill rats. Main toxins are brodifacoum ,Chlorophacinone ,Coumachlor ,Warfarin. The major manifestations of this toxin are coagulation dysfunction and liver cell failure.renal failre is very rare and is always due to severe hypotension(internal bleeding) or DIC. Rattol containing aluminum phosphate is also reported.

BRAKE OIL POISONING (ETHYLENE GLYCOL)

Brake fluid is a hydraulic fluid used in most brake applications for the vehicles. DOT 3 contains 40% of triethylene glycol monobutryl ether & 30% Diethylene glycol monobutryl ether.Once ingested, it is rapidly absorbed from gut. DEG is oxidised by alcohol dehydrogenase to 2 Hydroxy ethoxy acetaldehyde & then via aldehyde dehydrogenase to 2 hydroxy ethoxy acetic acid (HEAA). The

metabolic acidosis and organ dysfunction postulated to result from the generation of HEAA.The clinical features of DEG can be divided into 3 phases. First phase typically involves GI side effects like nausea, vomiting, abdominal pain and diarrhoea. Patient may have abnormal osmolal gap and anion gap metabolic acidosis. Second phase occurs 1-3 days following exposure, the hallmark is acute renal failure. ATN will be the usual biopsy picture. Multiple other effects reported are cardiac dysrhythmias, tachycardia, hypertension pulmonary edema and pancreatitis.Third phase occurs 1-2 weeks after the ingestion. Neurological complication dominates in this period. Peripheral neuropathy is a common occurrence and cranial nerve abnormalities including bilateral facial nerve palsy and bulbar palsy have been reported . Widespread denervation of limb muscles have been demonstrated. Patient may become quadriparetic and unresponsive, clinical course during this phase is unpredictable.

CORROSIVE POISONING

The corrosive poisoning is mostly accidental since it colour mimics water and soft drinks. When consumed accidentally the amount consumed will be small in quantity due to irritation produced by the corrosives in mouth. If it is consumed with suicidal intent(particularly under the influence of alcohol), then amount consumed will be high and systemic toxic symptom will develop.AKI after corrosive ingestion is not rare but direct damage to the kidney is seen only in severe poisoning. The factors that usually causes renal injury are hemodynamic instability, hemolysis, rhabdomyolysis, sepsis, severe acidosis and drugs. The risk factors the determine the

renal outcome are extremes of ages, amount consumed, time of presentation, type of corrosives and MODS.

MATERIALS AND METHODS

In this study we had selected all cases who had developed renal failure after toxin ingestion or animal bite (snake, wasp sting and scorpion). In this prospective study cases admitted to the Poison Control, Training and Research Centre of Government General Hospital, Madras Medical College were monitored and evaluated for development of AKI.

All the patients included in the study are subjected to Clinical history taking and complete physical examination.

Laboratory investigations included hemoglobin, total and differential leukocyte counts, platelet counts, red cell counts, bleeding and clotting time, coagulation profile including prothrombin time, activated partial thromboplastin time and international normalized ratio (INR), urine microscopy, urine albumin, kidney and liver function tests, and serum electrolytes.

Radiological investigations included X-ray chest and ultrasonography of abdomen. All patient were managed according to tamilnadu health system project guide lines. Anti snake venom protocol was strictly followed. All patients with snake bite was started on ASV as per TNHSP Protocol.

INCLUSION CRITERIA

All cases of toxin induced AKI (by AKIN criteria)are included in the study.

EXCLUSION CRITERIA

- Pre existing renal failure
- Features suggestive of CKD as per KDIGO definition
- Chronic NSAID intake
- Drugs that interact with creatinine estimation
- Tablet over dosage
- Drug induced renal failure

RESULTS AND OUTCOME

Total number of cases during the study period was 4125. Total number of death during the period was 264(6%). Total case presented with renal failure is 178(4.3%).Total number of cases with dialysis requirement is 130(83%).Total number of cases not requiring dialysis was 48 (17%).Total number of males is 115(64%)Total number of females 63(36%) .Total number of death in AKI IS 45(17%).most common cause of death is paraquat33%

causes	TOTAL		AKI	
Snake bite	915	22%	83	46.6%
Scorpion sting	152	3.5%	3	0.01%
Unknown bite	244	5.9%	0	0
Wasp	16	.03%	11	6.1%
Corrosives	433	10.4%	10	5.3%
Cuso4	13	.03%	5	2%
Paraquat	48	1%	27	15%
Rodenticide	420	10.1%	5	2.8%
Pesticide	1132	27.4%	15	8.4%
Plant poison	319	7.7%	9	5%
Alcohol	71	1.7%	1	0.05%
Hair dye	39	.5%	7	3.9%
Others	322	7.8%	2	1%
Crab bile	1	.001%	1	0.5%
Total	4125	100%	178(4.3%)	100%

Causes	TOTAL(M)	TOTAL(F)
Snake bite	54 (65%)	29(35%)
Wasp	9(82%)	2 (18%)
Scorpion	1(33%)	2(66%)
Paraquat	22(82%)	5(8%)
Hair dye	3 (42%)	4 (58%)
CuSO4	4 (80%)	1 (20%)
Insecticide	10 (66%)	5 (34%)
Corrosive	6 (60%)	4 (40%)
Plant	3 (34%)	6 (66%)
Ratkiller	2 (40%)	3(60%)
DEG	1 (100%)	0
TOTAL	115 (64%)	63 (36%)

causes	Dialysis requiring(m)	Dialysis requiring (f)	Not requiring dialysis(m)	Not requiring dialysis(f)	Death
Snake bite	47	22	8	6	10(16%)
Wasp	7	2	0	0	2
Scorpion	0	0	1	2	1
Paraquat	20	5	0	0	14(33%)
Hair dye	1	1	2	3	1
CuSO4	2	0	2	1	1
Insecticide	6	4	3	2	8(18%)
Corrosive	3	2	3	2	5(
Plant	1	1	2	5	1
Ratkiller	0	3	2	0	2
DEG	1	0	0	0	0
Total	90	40	24	24	45

MODE OF DIALYSIS

TYPE	HEMODIALYSIS	AIPD	TOTAL DEATH	death
Snake bite	66	3	17	10
Wasp	9	0	2	2
Scorpion	0	0	04	1
Paraquat	22	5	21	14
Hair dye	2	1	4	1
CUSO4	1	1	2	1
Insecticide	4	7	162	8
Corrosive	1	4	18	5
Plant	1	1	16	1
Ratkiller	2	1	18	2
DEG	1	0	0	0
TOTAL	109 (83%)	23 (17%)	264 (83%)	45(17%)

OUTCOME:

causes	Total cases	Partial recovery	Biopsy	Death	On MHD
Snake bite	83	14	20	10	3
Wasp	11	1	5	2	1
Scorpion	3	0	0	1	0
Paraquat	27	0	0	11	0
Hair dye	7	0	0	1	0
CuSO4	5	0	0	1	0
Insecticide	15	2	0	9	0
Corrosive	10	0	0	4	0
Plant	9	0	0	1	0
Rat killer	5	0	0	1	0
DEG	1	1	1	0	1
TOTAL	178	16	27	45	5

SNAKE BITE

Total number of snake bite admitted was 913,of which 83 had developed acute kidney injury and were categorized using AKIN criteria 69 (83%) patients were received dialysis. Various factors and its impact on dialysis requirement, severity of renal failure, outcome are analyzed

Residence: On comparison between the rural and urban population regarding the outcome was statistically insignificant (P value 0.30).

Age: On analyzing the age factor, the most commonly affected population was between 30 – 50 years. But it was found that progression to CKD and death are more in age group more than 50 (P value < 0.05).

Sex: On comparison 47 male and 22 females required dialysis and 8 from each group doesn't required dialysis. On detail analysis of the outcome it was found that 5 patients from each group progressed to CKD whereas 6 male and 4 female patients expired. On determining the severity and outcome of the patients by chi-square testing (p 0.45) , was found to no statistical significance.

Time of Bite: 50% of the bite occurred in the early morning from 4am to 8 am and 32% of bite occurred in the midnight and only 18% of bites were reported during day time. Maximum number of death i.e. 50% occurred in the early morning and it was statistically significant with P value < 0.05.

Type of Snakes: On analyzing the data, it was found that all three forms of viper bite shows significant statistical effect on both severity of renal of failure(P VALVE <0.003) and outcome (p.000).this is in concordance with Muthusethupathi et al, and Joseph K Joseph et al study.

Site of bite: 65% of bite occurred in lower limb, 30% of bite occurred in upper limb and 5% in trunk. On analyzing the severity and outcome, the site of bite has no

statistical significance. But study by Udaya Kumar et al in 2004 showed that bite in site with poor vascularity has least toxic.

Initial care: Of the 11 patients who had native medications 3 has expired and 7 had progress to CKD. But on comparing the primary, secondary and tertiary care treatment centers there is no significant change in the severity and outcome. This is probably due to better health care delivery and proper guideline based management protocol (by TNHSP) even at the primary care level itself.

Delayed Presentation: In a group of Seven patients whose diagnosis was made only by the treating physician are all dialysis dependent at presentation ($p<0.05$), but mortality was not statistically significant.

Time lag for referral: The time lag for referral in dialysis requiring group is 6.6 hours whereas who doesn't require dialysis is 5.3 hours. But the time lag for referral (according to t test for equality of needs) has no significant effect on severity of renal failure and outcome.

Time lag for ASV: In dialysis requiring group the mean time lag for initiating ASV is 9.94 hours whereas the patient who doesn't require dialysis the mean time lag if 7.5 hours. According to test for equality of variance it was statistically significant.

Dosage of ASV: 63 patients have completed ASV before the onset of renal failure (Group A). 20 patients had not completed ASV before the onset of renal failure (Group B). on analysis in group A 11% of patients expired and 14% of patients progressed to CKD. In Group B 15% of patients expired and 20% of patients

progressed to CKD.On applying chi square test it was found to have statistically significant with p value – 0.001.This is in concordance with Muthusethupathi et al, and Sitprija et al et al study.

Time of Completion of ASV: The patients are grouped into 3 categories based on completion of ASV , group A < 24 hrs, group B 24 - 48 hours and group C > 48 hours. Group A consists of 43 patients; Group B consists of 38 patients and 2 patients in Group C. On statistical analysis there is no significance in dialysis requirement and severity of renal failure. But out come (death and progression to CKD) for statistically significant in group B and C according to chi square test(p valve < .005).

Cellulitis: Patients were grouped into A and B, with and without cellulites. Group A consist of 69 patients of which 80.7% are dialysis dependent where as group B consist of 14 patients with 19% dialysis dependent. And regarding the outcome 9 patients in group A progressed to CKD and 10 expired, Where as in group B only one patient progress to CKD and no death reported. On statistical analysis with chi square test it was found that severity of renal failure and outcome are statistically significant. This observation is concordant with udayakuamr etal 2004 study.

Cellulitis	Total	Dialysis	Non	Recovered	Partial	Death
YES	69	57(85%)	12(75%)	50(79%0	9(90%)	10
NO	14	10(15%)	4(25%)	13(21%)	1(10%)	0

There is no statistical significance for dialysis requirement but p < 0.03 for outcome

Lymphadenopathy: Lymphadenopathy was present in 46 patients. On statistical analysis there was no significance either in the form of severity of renal failure or outcome.

Fang Marks: 23 patients had fang marks and are categoriesed under group A. It was found that dialysis requirement in group A was 95.7% when compared to 75% in group B. According to Pearson chi square formula it was statistically significant with P value of < 0.001. In the same group A 4 patients expired and 4 patients progress to CKD when compared to 6 patients in group B and these was also found to be statistically significant with P value of < 0.001. So presence of fang mark indicates furious and deep bite, which causes maximum envenomation.

	Total	**Dialysis**	**Non**	**Recovered**	**Partial**	**Death**
Yes	23	22	1	15	4	4
No	60	45	15	48	6	6
P valve		.034				.0005

On analysis presence of flang marks had significant correlation with severity and out come

DISTRIBUTION OF CASES:

Age	Total	DIALYSIS	CKD	DEATH
12 -20	2	2	NIL	NIL
21-30	14	12	1	1
31-40	19	14	NIL	1
41-50	25	20	3	4
51-60	15	13	3	4
ABOVE 60	8	6	3	1

Most of the bites are seen in middle aged men.(30-50).But outcome is severe ,more deaths and progression to CKD noted in age >50

RURAL VS URBAN

PLACE	TOTAL	DIALYSIS	DEATH
RURAL	60	48	5
URBAN	23	19	4

There is not much difference in out come in both group.

SEX DIFFERENCE

Sex	Dialysis requiring	Independent	Recovered	Partial	Death
Male	47(70%)	8(50%)	44(70%)	5(50%)	6(60%)
Female	20(30%)	8(50%)	19(30%)	5(50%)	4(40%)

Analysis revealed no statistical significance of sex with dialysis requirement and severity of renal failure.

TIMING OF BITE

Time	Total	Dialysis	Death
4 am -8am	42	31	6 (60%)
8am-6pm	14	14	1 (10%)
6pm-4 am	27	22	3 (30%)

Early morning bites has statistical significance in outcome ($p<.04$)

TYPE OF SNAKE AND OUTCOME

There is significance correlation of dialysis requirement with viper bite P VALVE <0.003 after eliminating unknown species

Type of snake	number	Dialysis requirement	Partial recovery	death
cobra	5(6%)	5	0	1
krait	4(4.8%)	2	0	0
Not known	30(36.1%)	25	2	2
Pit viper	3(3.6%)	3	3	3
Russell viper	31(37.3)	23	4	4
Saw scaled	10(12%)	9	4	0

Analysis shows there is a significant correlation with the type of snake

SITE OF BITE AND INTENSITY OF RENAL FAILURE

Site of bite	Total	Dialysis	CKD	Death
Upperlimb	25	21	4	4
Lowerlimb	54	42	6	5
Trunk	3	3	0	1

No statisticaal significance was found in between the groups

OUTCOME IN VARIOUS TREATMENT CENTERS

First care	total	Dialysis	Death	CKD
Primary	10	8	1	1
Secondary	41	32	4	3
tertiary	21	17	2	2
native	11	10	3	7

No statistical significance found except for native drug intake

DELAYED DIAGNOSIS AND OUTCOME

Diagnosed by	Total	Dialysis	Death	CKD
SELF	76	60	9	9
PHYSICIAN	6	7	1	2

Statistical significance found p valve <.05

TIME LAG AND OUTCOME= ANOVA-Time Lag for ASV

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	235.605	2	117.802	2.580	.002
Within Groups	3653.070	80	45.663		
Total	3888.675	82			

Time lag for ASV is statistically significant association with dialysis dependent group

ASV DOSAGE AND OUTCOME

COMPLETED ASV BEFORE	TOTAL	DIALYSIS	DEATH	CKD
YES	63	48	7	9
NO	20	18	3	4

Those in whom ASV is completed had a better outcome(p 0.04)

TIME OF COMPLETION OF ASV AND OUTCOME

TIME OF COMPLETION OF ASV	TOTAL	DIALYSIS	DEATH	CKD
<24	43 (52%)	31 (50%)	3(30%)	2 (72%)
24-48	38 (45%)	34 (47%)	5 (50%)	10 (14%)
>48	2 (3%)	2 (3%)	2 (20%)	2 (14%)

There is significant correlation with delayed ASV and renal outcome p valve < .005

ANALYSIS OF CLINICAL FACTORS

MODE OF PRESENTATION

	Local	Hematological	Neurological	Renal
Russell	28	12	1	12
Sawscaled	9	6	0	7
Pit viper	3	3	0	3
Cobra	4	0	4	1
Krait	0	0	4	0
unknown	28	8	2	9

RENAL FAILURE AT PRESENTATION AND OUTCOME

Cases	death	Partial recovery	Complete recovery
32	7	12	13

Of the 83 patients, 32(38%) had renal failure at the time of presentation. In these group 7(21%) patients died and 12(37.5%) progress to CKD. On comparing these group with remaining 51(62%) patients, those who had renal failure at the time of presentation has very poor outcome (P value 0.004).

CLINICAL FEATURES AND OUT COME

	total	**Dialysis dependency**	**death**	**Complete**	**Partial**
Edema	72	60	10	51	11
Lymph node					
Subconjuctival bleed	15	15	4	3	8
Spontaneous bleed	4	4	2	0	2
Breathlessness	20	19	5	11	4
Uremic symptoms	12	12	5	0	7
Oliguria	22	22	5	9	8

Presence of uremic symptoms: Of that 83 patients, 12(14.5%) patients had uremic symptoms at the time of presentation. All of them were dialysis dependent, 5 patients were expired and 7 patients progress to CKD.

LAB PARAMETERS AND OUTCOME

Parameters	Total	Dialysis	Complete	Partial	Death
Hematuria	13	13	2	7	4
Proteinuria	9	9	3	1	5
Anemia	19	17	6	8	5
Thrombocytopenia	9	9	1	4	4
Fragmented RBC	9	9	1	3	6
PT /APTT	43	41	22	11	8
U.MYOGLOBULIN	8	8	1	4	3

Proteinuria :Of the 83 patients, 9(10%)had proteinuria at the time of presentation. All of them were dialysis dependent, 5 patients were expired and 2 patients progress to CKD.

Thrombocytopenia: Of the 83 patients, 9(10.5%) had thrombocytopenia at the time of presentation. All of them were dialysis dependent, 4 patients were expired and 4 progressed to CKD, 2 patients was diagnosed to have thrombotic micro angiopathy in biopsy.

Fragmented RBC: Of the 83 patients, 9(10.5%) had fragmented RBC. All of them were dialysis dependent, 6 patients were expired and 3 patients progress to CKD, 2 patients was diagnosed to have thrombotic micro angiopathy in biopsy.

Subconjunctival haemorrhage : Of the 83 patients, 15(18%) had subconjectival haemorrhage during the course of illness. These patients were dialysis dependent and 4 patients expired and 8 patients progress to CKD. On comparing these group with remaining 68(82%) patients, those who had suconjunctival haemorrhage at the time of presentation has very poor outcome.($P < 0.04$)

Spontaneous bleeding :Spontaneous bleeding was noted in 4 out of 83 patients, of which two had expired and two progressed to CKD. All the 4 had signs of DIC. So the presence severe hematological manifestation has significant role in renal outcome.

Prolonged PT, APTT: Of the 83 patients, 43(51%) had prolonged PT, APTT. 41 of them were dialysis dependent, 8(80%) patients were expired and 11(78%) patients progress to CKD.

Urine myoglobulin : Of the 83 patients, 8(10%) had urine myoglobulin. All of them were dialysis dependent: 3 patients were expired and 4 patients progress to CKD.

Whole Blood Clotting time :

WBCT	Total	Dialysis	Non	Recovered	Partial	Death
<12	30	23(35%)	7(44%)	27(43%)	2(20%)	1(10%)
12-24	25	18(27%)	7(44%)	19(30%	2(20%)	4(40%)
24-36	18	16(24%)	2(12%)	12(19%)	5(40%)	1(10%)
>36	10	10(14%)	0	5(8%)	5(40%)	4(40%)
P		<0.05				<0.05

By using the chi square test in whole blood time normalization has high statistical significance in association with outcome and recovery p value < 0.004.

RENAL PRESENTATION AND DIALYSIS REQUIREMENT

	AKIN I	**AKIN II**	**AKIN III**
YES	1	3	63
NO	3	12	1

Parameter	**Dialysis requiring**	**Not requiring dialysis**
Presenting sr.cr	2.0377	2.0325
Peak sr.cr	7.3	2.3
Range	9.3 -0.6	0.7 -3.4
Hypercatabolic	27	0
Mean time for renal failure	35.5	43.5
Mean time for ASV completion	27.5	23.5
Death	10	Nil

As mentioned in the above table, mean presenting serum creatinie is almost same. But peak value is 7.3 for dialysis dependent group. All the cases of hyper catabolic aki are dialysis dependent. Mean time of onset of renal failure is much earlier in the first group and was found to be statisticaly significant. Mean time for

completion of ASV was more or same and has significant difference. All the death has been reported only in the dialysis requiring group. Twenty patients underwent renal biopsy and fourteen patients had only partial recovery with three patients became dialysis dependent(on MHD).

HYPERCATABOLIC AKI: 27(32.5%) patients had satisfied the criteria for hyper catabolic AKI. Of which 16 are male and 11 are female. And following were some important features associated with this form of AKI .Totally 9(30%) patients received native treatment (statistically significant). 10 cases are due to Russell viper, 5 due to saw scaled, 3 due to pit viper and 1 due to cobra and remaining 8 species not known.

Mean time of presentation: Day1 - 8 , Day2 - 17 and Day3- one patients.ASV was not completed in 21(77%) patients. Cellulitis was present in 25(92%) patients. Lymphadnopathy was present in 17(62%) of patients .Fang marks are present in 9(34%) of patients.

Renal failure at the time of presentation in 13(50%) of patients. Urine showed myogloblin in 7 (25%) of patients. Jaundice was present in 9(34%) of patients. DIC was reported in 11(42%) of 27 patients.

Type	HISTOLOGY	OUTCOME(eGFR)	FOLLOW UP
Saw scaled	ATN	Recovered	Recovered
Saw scaled	ACN	82	CKD 3
Sawscaled	ATN	72	CKD3
Sawscaled	ACN	86	CKD4
Sawscaled	ATIN	76	CKD3
Sawscaled	ATN	45	ON MHD
Rusell	ATN	Recovered	Recovered
Rusell	ACN	60	CKD4
Rusell	ACN	56	CKD3
Rusell	TMA	72	EXPIRED
Rusell	TMA	Expired	Expired
Not known	ATN	25	ON MHD
Notknown	ATN	45	ON MHD
Not known	ACN	82	CKD4
Notknown	ATIN	Expired	Expired
Not known	ACN	78	CKD 3
Not known	ATIN	Recovered	Recovered
Not known	ATN	Recovered	Recovered
Not known	ATN	Recovered	Recovered

Twenty patients underwent renal biopsy and fourteen patients had only partial recovery with three patients became dialysis dependent (on MHD).In the biopsy there is no specific correlation with type of snake bite, except for two cases of TMA in Russell viper. Other histological features in biopsy are

ATN; of the eight cases (40%) reported, four case had been recovered. One case became CKD 3A, three other remain dialysis dependent. But no death was reported.

ATIN: Three cases had been reported and steroid was started. Of the three one patient died of sepsis (steroid was withdrawn due to CRBSI), one became CKD3A and one completely recovered.

TMA: Both case expired after becoming dialysis dependent.

ACUTE CORTICAL NECROSIS: Six cases have been reported. Four of them with patchy cortical necrosis and two had focal necrosis. All six had became dialysis independent. At last follow up three were in CKD stage 4 and three were in stage 3A.

Death: Total death observed is ten (12.5%).Cause of death includes DIC in 4(40%), sepsis 5(50%), VAP 2(20%). Type of snake is pit viper in 3 patients, Russell in 4 patients, Not known in one and cobra in one. The result was statistically significant by chi-square analysis.

PARAQUAT

Total numbers of cases are 27.More common in middle age men. The most common age group is 2nd and 3rd decade .Male is to female ratio is 5:1.19(70%) patients are illiterate and 12(44%) patients are under the influence of alcohol.

Among the individual who consumed under influence of alcohol 9(65%) expired, and was found to be statistically significant (P<0.04) .Average time to initial presentation is 4 hours (range 1-88 hours) .

Pre emptive dialysis was done in 10 patients .Average time lag to pre emptive dialysis is 22 hours .But presumptive hemodialysis had no statistical significance ,this indicates delayed presentation and need for hemofiltration. The consumption of paraquat was detected by the attenders in 12 cases (for which the average period of delay was 19 hours) .This delay in diagnosis led to mortality in 75 % of this group.

On multivariate analysis time lag for diagnosis, time lag for hemodialysis and presence of anemia has no statistical significance.

We had categorized the patient into four groups based on the amount consumed(Group A >50ml,Group B 50-30ml,GroupcC 30-10ml,and Group D<10ml).

Parameters	>50ml (A)	50-30ml (B)	30-10ml (C)	<10 ml(D)
Number	6	7	11	3
Alcohol	5	4	5	0
Time lag hrs	2	2.3	3	6
Total lag (HD)	21	22	33	19
Preemptive	4	3	3	3
Hb >9	6	5	7	2
Sr.cr (p)	2.68	2.59	2.86	1.7
Total hd	3.5	6.5	3	2
Duration	3.4	5	5	3
ARDS	5	4	5	0
PD	2	2	1	0
Death	6	5	3	0
Hepatitis	4	3	4	0
Cardiac inv.	0	1	1	0
Mean time death	4.23	5.4	5.5	0

In GROUP A(6) ,presenting mean serumcreatinine is 2.68,total patients given HD is 3 and PD in 3(50%). Average session of HD required is 3.5,for mean period of 3.4 days. Multi organ involvement is seen in 4(66%) and ARDS in 5(83.3%).Death occurred invariably in all patient with mean time 4 days.

In GROUP B(7) ,presenting mean sr. creatinine is 2.3,total patients given HD is 5 and PD in 2 (33%). Average session of HD required is 6.5,for a mean period of 5 days.Multi organ involvement is seen in 3(40%) and ARDS in 4(57%).Death occurred in 5(71%) with a mean time of 5.4 days.

In GROUP C(11) ,presenting mean sr.creatinine is 2.86,total patients given HD is 9 and PD in 2 (22%). Average session of HD required is 3,for a mean period of 5 days. Multi organ involvement is seen in 4(35%) and ARDS in 5(45%).Death occurred in 3(27%) with a mean time of 5.5 days.

In GROUP D(3) ,presenting mean sr.creatinine is 1.7 ,total patients given HD is 2 and PD in 2 (22%). Average session of HD required is 2,for a mean period of 3 days with no Multi organ involvement, ARDS or Death. On analysis it was found that amount consumed is the most important factor for multi organ involvement, renal failure (**p valve is <.003)**and death (**p valve is <.003)** Preeumptive hemodialysis has not proven to be significant role. By chi square analysis hemogloglin <9 had statistically significant outcome

HYMENOPTERA BITES

Total cases reported is 16, and 11 (%) presented with AKI. On which 9(81%) patients are male and 2(19%) are female. Mean age of presentation is 42.9.More common in lower socioeconomic group Number of stings ranges from 34 -88 but on analysis by t test number of stings has no impact on outcome Sex has significant effect on outcome p<0.45.Two patients received native medication and was found to be statistically significant. (p valve of 0.001,which is highly significant).

Plasma HB was detectable in 2 patients and by statistical analysis pearson chi-square test p valve is<0.001.Average referral lag in patient who died is 15.5 when compared to 5.5 hrs in remaining and has definite statistical significance p valve <.005 by levene's test for equality of variance.

Rhabdomyolysis was seen in82% of patients, Total number of FAD given was 11.Mean peak sr.cr is 7.6.Indication for hemodialysis was 6 with rhabhdomyolysis,3 with acidosis, 3 with both. Totally nine(82%) required hemodialysis. Average session is 10 (ranges 2-30)and average duration is 18 days (2-33).

Two patients died one due to rhabdomyolysis and next patient (defaulted on steroids) acidosis .Of the 9 patients with rhabdomyolysis, 2 patients developed ATN,2 developed AIN and two died. Of the eleven patients 6(57%) patients presented with hyper catabolic AKI.Proteinuria was in all 2 patients with ATIN.

Biopsy was done in 4 patients

BIOPSY	ATN	ATN/AIN	AIN
MALE	1(myoglobulin pigment)	1	1
FEMALE		1	

(Steroids was given in 3 patients with AIN, 2 recovered and one last follow up and became CKD).The intensity of renal failure is much severe (renal failure requiring dialysis), particularly for prolonged period. And early biopsy is always

needed in suspicion of AIN, which is curable with steroids. Cause of death in most occasions will be due anaphylaxis, rhabdomyolysis and renal failure

Plasma HB and Outcome

Plasma HB	total	recovered	Death
YES	2	0	2
NO	9	9	0
P value			**<0.001**

SCORPION

	TOTAL	**DIALYSIS**	**DEATH**	**CAUSE**
MALE	1	NO	0	NIL
FEMALE	2	NO	1	CARDIOGENIC SHOCK

In our study period we 152 cases admitted with scorpion sting ,but only 3 had renal failure. none of the three required dialysis . Average peak sr.cretinine 2.1All three cases belong to AKIN II Death was noted in 17 year old girl with autonomic imbalance.

HAIR DYE POISONING

Total patients with poisoning is 39.Total males are 3 and female are 4.Mean age of presentation 26.Total number of AKI is 7.FAD done in six cases. Dialysis

requiring renal failure is in two cases.Rhabdhomyolysis seen in 6 cases. Since toxicity depends on amount consumed we had grouped the patients into three

Group A : (Those who had consumed >50 ml) Only one patient in this group who presented with angioedema, rhabdomyolysis, peak s.cretinine of 6.7,evidence of hemolysis died on day of admission.

Group B : (Those who had consumed 25-50 ml.) Total 3 patients were in this group all 3 presented with rhabdomyolysis, mean peak creatinine2.9.one patient received hemolysis and the other two received FAD.

Group C: (Those who had consumed 25-50 ml.) Total 3 patients were in this group all 3 presented with rhabdomyolysis, mean peak creatinine1.7 .All three received FAD.

COPPER SULPHATE POISONING

PARAMETER	MALE	FEMALE
NUMBER	3	2
FAD	2	1
HD	0	1
PD	1	0

Cause of AKI	Hemolysis	Hemodynamic	Not known
Recovered	1	0	2
Death	1	1	0

One patient died of arrhythmia and one died due hemodynamic instability

RAT KILLER POISONING

TYPE	MALE	FEMALE
RATTOL	1	2
COMARIN DERIVATIVE	1	0
ZINC PHOSPHIDE	0	1

TYPE	HD	PD
RATTOL	2	0
COMARIN DERIVATIVE	0	0
ZINC PHOSPHIDE	0	1

Both female with rattol poisoning was expired due to fulminant hepatitis

PLANT POISONING

TYPE	MALE	FEMALE	DIALYSIS	NOT NEEDED	DEATH
OLEANDER	2	1	1	1	0
ODUVNTHALAI	1	5	1(PD)	5	1

Parameters	Oleander	Oduvanthalai
Number	3	6
Mean age	24	26
Mean peak creatinine	1.9	2.2
PD		1
HD	1	0
Recovered	3	5
Death	0	1 severe hyperkalemia

INSECTICIDE

Total number of AKI is 15 and total death is eight. All the eight patients who died are ventilator dependent and six of them had hemodynamic instability

DAYS OF ONSET OF AKI	HD	PD	DEATH IN HD	DEATH IN PD
<7	3	1	1	1
7-14	1	3	1	3
>14	0	2	0	2

	Male	Female	HD	PD	DEATH	On ventilator	Hemodynamic
OPC	4	3	2	4	4	5	4
Cell oil	0	1	1	0	1	1	1
Karate	1	1	0	1	1	1	1
Craine killer	1	0	0	0	0	0	0
Carbamate	1	0	0	0	0	0	0
Quinophos	1	0	1	0	1	1	0
Unknown	1	1	0	1	1	0	1

CORROSIVE POISONING

SEX	NUMBER	HD	PD	NON DIALYSIS	DEATH
MALE	6	1	2	3	3
FEMALE	4	0	2	2	2

Indication for dialysis is severe acidosis in 4 patients and anuria in one patient. Cause of death was sepsis and hemodynamic instability

DISCUSSION

Total Number of poisoning cases admitted in our unit during the study period was 4125.Total number of acute kidney injury according to AKIN classification is 178 (4.3%).Of them males are 115(64%) and females are 63(36%). Number of cases that required dialysis was 130 of which 21(17%) received Acute intermittent peritoneal dialysis due to hemodynamic instability and the remaining received HD (83%).

Renal biopsy was done in 20 patients with snake bite who are dialysis dependent for more than fourteen days, 5 patients with wasp sting, one patient with crab fish bile and one with brake oil poisoning.

Total number of death due to poisoning is 131, of which death in toxin induced AKI was 23 (17%).On comparisons with study conducted by the same institute in 2007-2009 where 32 cases developed ARF. 24 were due to snake bite, the rest due to chemical poisons.

SNAKE BITE

Total number of snake bite admitted was 913, of which 83(9%) had developed acute kidney injury and were categorized with AKIN criteria, 69 (83%) patients received dialysis. Among the toxin induced AKI incidence of snake bite induced AKI is 46.6%.

Various factors and its impact on dialysis requirement, severity of renal failure, outcomes were analyzed. On comparison between the rural and urban population regarding the outcome was statistically insignificant (P value 0.30).

On analyzing the age factor, the most commonly affected population was between 30 – 50 years. But it was found that progression to CKD and death are more in age group more than 50 (P value < 0.05) as was observed by Attappan et al.

On determining the severity and outcome of the patients by chi-square test (p 0.45), was found to be of no statistical significance. As stated by Muthusethupathi et al increased incidence in males is due to increased exposure

50% of the bites occurred in the early morning from 4Am to 10 Am and 32% of bites occurred in the midnight and only 18% of bites were reported during day time. Maximum number of deaths i.e. 50% occurred in the early morning and it was statistically significant with P value < 0.05.Patil TB et al showed incidence during early morning and during farm working hours.

On analyzing the data, it was found that all three forms of viper bite shows significant statistical effect on both severity of renal of failure(P <0.003) and outcome (p.000) and this is in concordance with Muthusethupathi et al, and Joseph K Joseph et al study. Incidence is more with Russell viper and mortality more in pit viper

65% of bite occurred in lower limb, 30% of bite occurred in upper limb and 5% in trunk. On analyzing the severity and outcome, the site of bite has no statistical

significance. But study by Udaya kumar et al in 2004 showed that bite at site with poor vascularity has least toxic.

On comparing the primary, secondary and tertiary care treatment centers there is no significant change in the severity and outcome. This is probably due to better health care delivery and proper guideline based management protocol (by TNHSP) even at the primary care level itself.

The time lag for referral in dialysis requiring group was 6.6 hours whereas who did not require dialysis was 5.3 hours. But the time lag for referral (according to t test for equality of needs) has no significant effect on severity of renal failure and outcome. But earlier studies by Attappan et al and Patil et al showed statistical significance. This may be due to proper primary care and early referral

Time lag for administration of ASV according to test for equality of variance was statistically significant with severity of renal failure and outcome. Those who completed ASV before the onset of renal failure was found to have statistically significant with p value – 0.001.This is in concordance with Udayakumar et al, and Sitprija et al et al study.On statistical analysis there is no significance in dialysis requirement and severity of renal failure in those who completed ASV after 24 hrs. But out come(death and progression to CKD) was statistically significant .

It was found that severity of renal failure and outcome are statistically significant in those with cellulites as observed by Udayakuamr etal 2004 study. In our study presence of lymph adenopathy has no significance either in the form of severity of renal failure or outcome. But according to Attappan et al regional

lymphadenopthy was another significant independent factor for ARF. Just as cellulites, regional lymphadenopthy can be a clinical indicator of the amount of toxin released by the snakebite.

Presence of fang mark indicate furious and deep bite, which causes maximum envenomation and was highly significant with relation to dialysis requirement, progression to CKD and death. Those who had renal failure and uremic symptoms at the time of presentation had very poor outcome.

Those who had proteinuria, hematuria, anemia, and thrombocytopenia are associated with poor outcome. Patients who had signs of DIC were associated with viper bites and presence of DIC is an important factor prognosticating factor as stated by Muthusethupathy et al, Joseph K Joseph et al and Attapan et al. In our study, 4 patients developed bleeding, which was less than that in other reports. All the 4 had signs of DIC. So the presence of severe hematological manifestations had significant role in renal outcome. Spontaneous bleeding was noted in 4 out of 83 patients, of which two had expired and two progressed to CKD. According to Muthusethupathi et al hemorrhage is the major symptom of systemic viper poisoning,Persistent bleeding from fang wounds, venepuncture sites .

In our study 43(51%) had prolonged PT, APTT. 41 of them were dialysis dependent, 8(80%) patients expired and 11(78%) patients progress to CKD. Of the 83 patients, 8(10%) had myoglobulinuria. All of them were dialysis dependent: 3 patients expired and 4 patients progressed to CKD. Whole blood time normalization has high statistical significance in association with outcome and recovery p value <

0.004.those with early normalization of WBCT was associated with reduced dialysis requirement and improved outcome

.27(32.5%) patients had satisfied the criteria for hyper catabolic AKI. Of which 16 are male and 11 are female. They are mostly observed in viper bite, native medication intake, patient with cellulitis, lymphadenopathy, flang marks. Most of them had renal failure at the time of presentation and associated with DIC.

On analyzing the renal parameters the is not much difference in presenting serum creatinine, but the average peak sr.creatinine, mean time for onset of renal failure, and the mean time for completion of ASV were significantly high.

On analyzing the histopathology there was no specific correlation with type of snake bite, except for two cases of TMA in Russell viper. ATN accounts for Eight cases (40%) ,ATIN in three cases(15%) ,TMA in two cases(10%),cortical necrosis in Six cases(30%) .Total death observed is ten (12.5%).cause of death includes DIC in 4(40%), sepsis 5(50%), VAP 2(20%).Type of snake is pit viper in 3 patients, Russell in 4 patients, Not known in one and cobra in one. The result was statistically significant by chi-square analysis.

According to Patil TB et al mortality of snake bite-induced acute renal failure is found to be 15.5% in this study. Kalantri *et al* reported an overall mortality of 11% in venomous snake bite patients. In our study mortality is (12.5%)

PARAQUAT

The most common age group is 2nd and 3rd decade .Male is to female ratio is 5:1.Among the individual who consumed under influence of alcohol 9(65%) expired, and was found to be statistically significant (P<0.04) .

Average time to initial presentation is 4 hours (range 1-88 hours) .Pre emptive dialysis was done in 10 patients .Average time lag to pre emptive dialysis is 22 hours .But presumptive hemodialysis had no statistical significance ,this indicates delayed presentation and need for hemofiltration. The delay in diagnosis led to mortality in 75 % of this group.

On multivariate analysis time lag for diagnosis, time lag for hemodialysis and presence of anemia has no statistical significance in outcome. Total number of death is 14(55%),in that 11 have consumed more than 50 ml.

On analysis it was found that amount consumed is the most important factor for multi organ involvement ,renal failure (p valve is <.003)and death (p valve is <.003)**.**This results are well supported by study done by *JS Sandhu et al,who showed* that severity of poisoning, late referral and MODS showed increased mortality.

HYMENOPTERA BITES

Total cases reported is 16, and 11 (%) presented with AKI. On which 9(81%) patients are male and 2(19%) are female. As it was mentioned in previous studies Mejia Velez et al, though hymenoptera is a rare cause of animal bite ,it causes higher percentage of AKI .Mean age of presentation is 42.9.

Number of stings ranges from 34 -88 but on analysis by t test number of stings has no impact on outcome. Sex has significant effect on outcome p<0.45.Plasma HB was detectable in 2 patients and by statistical analysis pearson chi-square test p valve s<0.001.Average referral lag in patient who died is 15.5 when compared to 5.5 hrs in remaining and has definite statistical significance p valve <.005 by levene's test for equality of variance.

Rhabdomyolysis was seen in82% of patients in our study, which is supported by Zhang ling, Tang yi et al study. Mean peak sr.cr is 7.6.Totally nine(82%) required hemodialysis for an average duration is 18 days (2-33). The intensity of renal failure is much severe renal failure requiring dialysis, particularly for prolonged period as evidenced by sitprija et al.

Early biopsy was done in 4 patients ,1 patient had ATN with myoglobin pigments,1 with ATN with AIN, and 1 developed AIN .Steroids was given in 3 AIN, 2 recovered and one last follow up and became CKD .Early biopsy is always needed in suspicion of AIN, which is curable with steroids supported by.

Two patients died one due to rhabdomyolysis and next patient (defaulted on steroids) acidosis. Cause of death in most occasions will be due anaphylaxis, rhabdomyolysis and renal failure supported by Zhang ling, Tang Yiet al study

SCORPION

Scorpion bite is very common in Tamilnadu. But the incidence of AKI scorpion bite rarely reported .in our study period we 152 cases admitted with

scorpoin sting , but only 3 had renal failure. None of the three required dialysis . Average peak sr.cretinine 2.1and all three cases belong to AKIN II .Death was noted in 17 year old girl with autonomic imbalance.

HAIR DYE (SUPERVASMOL) POISONING

Total patients with poisoning is 39 of whom AKI was seen in 7(18%).Total males are 3 and female are 4.Mean age of presentation was 26yrs.Dialysis requiring renal failure is seen in two cases and Rhabdomyolysis seen in 6 cases. Since toxicity depends on amount consumed we had grouped the patients into three, of which

Patient who had consumed >50 ml was presented with angioedema, rhabdomyolysis, evidence of hemolysis died on day of admission .Since the same size is too small, statistical analysis is not done. In a retrospective study which included 81 consecutive patients of self poisoning with Supervasmol 33 ®, who SVIMS, Tirupati, between May 2008 and April 2010. suggest that the ingestion of more than 50ml of the dye was associated with significant rhabdomyolysis and acute renal failure, which required haemodialysis.

COPPER SULPHATE POISONING

The trend of these poisoning is coming done ,probably due to reduced availability.

Total 13 case were received in the study period and total no. of AKI was 5(40%).

One female patient was dialysis depedent and one male patient received peritoneal dialysis both the two patient had evidence of hemolysis. One patient died of arrhythmia and one died due hemodynamic instability.In a study done by chug et al eleven out of a series of twenty-nine patients (37-9%) with acute copper sulphate poisoning developed acute renal failure. Intravascular haemolysis appeared to be the chief factor responsible for renal lesions in these patients.

RAT KILLER POISONING

Only five cases reported with AKI, 3 patients affected by rattol(aluminium phosphide),one with warfarin derivative and one with zinc phosphide. Three patients required dialysis and one was given AIPD. Both female with rattol poisoning was expired due to fulminant hepatitis.

PLANT POISONING

Total nine cases are reported of which six where due to oduvanthalai and three due to oleander. Four patients received AIPD and one oduvanthalai required dialysis. Renal failure in all cases of oleander is due to cardiogenic shock where as oduvanthalai has direct nephrotoxic effect .one patient with oduvanthalai expired due to refractory hyperkalemia.In a study conducted by CMC Vellore ,it was postulated that renal failure, oliguric in most instances, has been documented across clinical studies, though its etiology is probably multifactorial: direct toxin effect secondary to hypotension and, possibly, secondary to hypokalemic rhabdomyolysis.

INSECTICIDE

Total number of AKI is 15 and total death is eight. HD was given in 4 patients and AIPD was done in six patients with hemodynamic instability. All the eight patients who died are ventilator dependent and six of them had hemodynamic instability.

CORROSIVE POISONING

Total ten patients presented with renal failure. Indication for dialysis is severe acidosis in 4 patients and anuria in one patient. Four patients received AIPD and one received hemodialysis.Six patients were expired because of sepsis and hemodynamic instability. According to Vivek et al corrosive induced renal failure is usually due to secondary effect

BRAKE OIL POSONING

One patient had who had consumed brake oil presented with clinical features like abdominal pain, vomiting, acute tubular necrosis, pancreatic injury, quadriparesis and transient VIII nerve involvement which improved during discharge.

CRAB BILE POISONING

One case was reported with ingestion of crab bile. He presented with dialysis dependent renal failure and was diagnosed as ATIN.

CONCLUSION

- Incidence of AKI in our study population was 178 (4.3%) of which dialysis requirement was seen in 130 (83%).
- Snake bite was the leading cause of toxin induced AKI (46%).
- Risk factors for the development of AKI in this population include cellulitis, regional lymphadenopathy, presence of fang marks, while predictors of poor outcome were hypotension, DIC and rhabdomyolysis.
- Early adequate administration of snake anti-venom had reduced the severity of AKI.
- Type of snake bite particularly pit viper bite had very poor outcome.
- Paraquat and wasp had very high incidence and mortality among the study group.
- Amount of paraquat consumed was a poor prognostic factor.
- Preemptive hemodialysis was found to be ineffective in our study population.
- In acute interstitial nephritis due to wasp, timely and appropriate management usually cures the patient without any residual damage.
- Insecticide, corrosive although high in number had a very low incidence of AKI but highly fatal due to associated multi organ involvement.
- Single case of brake oil poison was observed and presented as biopsy proven AIN

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MASTER CHART

name	age	AGE CODE	sex	place	education	timing	place of bite	site of bite	type of snake	first visit	native	diagnosed by physician	TIME LAG FOR REFERRAL	time lag for ASV
MOHAN	33	3	1	1	2	1	1	4	NOT KNOWN	3	NO	SELF	3	3
VACHITHANATHAM	38	3	1	1	1	1	1	4	PIT VIPER	2	NO	SELF	6	8
SUDAKAR	24	2	1	1	2	1	1	4	RUSELL	3	NO	SELF	4	5
MANI	32	3	2	1	1	1	2	4	RUSELL	3	NO	SELF	3	4
PERUMAL	45	4	1	1	1	3	1	4	SAW SCALLED	1	NO	SELF	4	5
POOVARAGAVAN	42	4	1	1	1	3	1	4	COBRA	2		SELF	2	3
PALANI	40	4	1	1	2	3	1	4	NOT KNOWN	3	NO	PHYSICIAN	12	12
MOORTHI	48	4	1	1	2	1	1	4	NOT KNOWN	2	NO	SELF	2	2
KANAGAMAL	54	5	2	2	1	1	2	2	PIT VIPER	4	LOCAL APPILICATION	SELF	4	9
NATARAJAN	42	4	1	2	1	2	1	4	KRAIT	2		SELF	3	3
NAGARAJAN	60	6	1	1	1	3	3	2	NOT KNOWN	2	NO	SELF	2	24
SWARAJKUMAR	45	4	1	2	3	3	3	4	RUSELL	1	NO	SELF	3	21
POVARASI	28	2	2	1	1	1	2	2	SAW SCALLED	2	NO	SELF	3	12
SHANKAR	20	2	1	1	1	1	1	4	RUSELL	4	ORAL DRUGS	SELF	14	15
111	64	6	2	1	1	1	1	4	SAW SCALLED	4	ORAL DRUGS	SELF	16	19
PARASURAM	38	3	1	1	1	2	1	2	RUSELL	2	NO	SELF	6	8
AMBROSE	44	4	1	1	1	3	1	4	NOT KNOWN	2	NO	SELF	4	4
SAKUNTHALA	52	5	2	1	2	1	1	2	RUSELL	2	NO	SELF	6	6

DEVENDRAN	47	4	1	1	1	1	1	4	RUSELL	2	NO	SELF	4	6
ANBALAGAN	43	4	1	1	2	3	1	2	RUSELL	4	ORAL DRUGS	SELF	25	26
SUBRAMANI	37	3	1	1	2	1	1	4	COBRA	2	NO	SELF	5	6
RAJI	45	4	1	1	3	1	1	4	RUSELL	2	NO	SELF	3	5
SARAVANAN	21	2	1	1	1	2	1	4	KRAIT	2	NO	SELF	5	6
ULAGANATHAN	50	5	1	1	1	3	1	4	RUSELL	1	NO	SELF	1	1
PATCHAIAMMAL	35	3	2	1	1	3	1	4	RUSELL	1	NO	SELF	2	3
LAKSHMI	42	4	2	2	1	3	2	2	PIT VIPER	3	NO	SELF	6	7
KAMATHCHI	45	4	2	1	1	1	1	2	NOT KNOWN	2	NO	PHYSICIAN	16	17
MUNNA	30	3	2	2	3	3	2	2	NOT KNOWN	2	NO	SELF	4	4
DEVARAJ	43	4	1	2	1	1	2	3	RUSELL	1	NO	SELF	3	21
PANDIAN	50	5	1	2	2	3	2	2	RUSELL	2	NO	SELF	6	8
KAMATHCI	60	6	2	1	1	1	3	2	SAW SCALLED	3	NO	SELF	3	4
MANJULA DEVI	20	2	2	1	3	2	3	2	NOT KNOWN	4	ORAL DRUGS	SELF	18	20
TAMINA	15	1	1	2	3	2	3	4	NOT KNOWN	2	NO	SELF	4	4
SUBRAMANI	59	6	2	1	2	3	1	4	SAW SCALLED	4	ORAL DRUGS	SELF	14	15
SENGAIYA	77	7	1	1	2	1	1	4	NOT KNOWN	1	NO	SELF	3	4
JAYARAMAN	28	2	1	1	1	1	3	4	NOT KNOWN	2	NO	SELF	7	8
RAJAMMAL	45	4	2	1	1	1	3	4	RUSELL	4	ORAL DRUGS	SELF	3	14
KANIYAMMAL	50	5	1	2	1	1	2	4	NOT KNOWN	2	NO	SELF	6	8
VARALAXMI	45	4	2	2	1	1	2	4	RUSELL	3	NO	SELF	4	5
SUDHAKAR	37	3	1	2	1	1	2	4	RUSELL	2	NO	SELF	4	5
PATCHAIAMMAL	33	3	2	1	1	3	1	3	SAW SCALLED	2	NO	SELF	4	5

VASNTHAMMAL	65	6	2	1	1	3	1	3	SAW SCALLED	4	ORAL DRUGS	SELF	23	25
SUBRAMANI	44	4	1	1	1	1	1	2	RUSELL	3	NO	SELF	4	5
SARAVANAN	33	3	2	1	1	1	1	2	NOT KNOWN	2	NO	YES	9	10
SAROJAMMAL	55	5	2	1	2	1	1	2	NOT KNOWN	2	NO	PHYSICIAN	4	4
SELVAM	35	3	1	1	2	2	3	2	RUSELL	2	NO	SELF	1	12
SARAVANAN	34	3	1	1	2	2	3	4	RUSELL	2	NO	SELF	6	8
KAVITHA	23	2	2	1	2	1	1	4	RUSELL	3	NO	SELF	1	2
KUMAR	32	3	1	1	2	3	1	2	NOT KNOWN	2	NO	SELF	4	4
DANALAKSMI	25	2	2	1	1	3	1	4	NOT KNOWN	2	NO	PHYSICIAN	4	4
JEYARAMAN	56	5	1	1	1	2	1	2	NOT KNOWN	2	NO	PHYSICIAN	16	24
MNUSWAMY	16	1	1	1	1	1	1	4	NOT KNOWN	3	NO	SELF	3	3
RAJI	33	3	2	2	1	1	2	2	RUSELL	3	NO	SELF	6	6
SUNDARAM	24	2	1	2	2	2	2	4	SAW SCALLED	1	NO	SELF	4	5
SEKAR	47	4	1	2	2	3	2	2	RUSELL	2	NO	SELF	12	12
SELVAM	50	5	1	2	2	3	2	4	COBRA	2	NO	SELF	3	3
SALAMMAL	55	5	1	2	1	2	3	4	COBRA	3	NO	SELF	5	5
SIVASHANKAR	45	4	2	1	1	1	1	4	KRAIT	2	NO	SELF	1	2
PARIMALA	40	4	2	1	3	1	1	4	RUSELL	2	NO	SELF	2	12
PALANI	24	2	1	1	2	1	1	4	NOT KNOWN	2	NO	SELF	4	4
MANIVANNAN	33	3	1	1	3	2	1	4	NOT KNOWN	3	NO	SELF	4	4
NAGAMMAL	54	5	2	2	1	3	2	4	NOT KNOWN	3	NO	SELF	3	3
KUPPAN	57	5	1	1	1	3	1	4	RUSELL	1	NO	SELF	4	5
KANDASWMY	45	4	1	1	1	1	1	2	RUSELL	1	NO	SELF	2	6
VARADHARAJAN	34	3	1	1	2	1	1	4	SAW SCALLED	2	NO	SELF	3	12
MAHENDRAN	40	4	1	2	3	1	1	4	RUSELL	2	NO	SELF	3	21

RAJAN	24	2	1	1	3	3	1	4	NOT KNOWN	3	NO	PHYSICIAN	24	26
NAGARAJAN	40	4	1	2	3	3	1	2	COBRA	4	TOURNIQUET	SELF	22	22
SHANKAR	24	2	1	1	3	2	1	2	RUSELL	2	NO	SELF	4	5
KAMATHCHI	29	2	1	1	1	2	3	4	NOT KNOWN	4	ORAL DRUGS	SELF	14	16
NAGARAJAN	60	6	1	1	2	3	3	4	NOT KNOWN	3	NO	SELF	14	18
MANAVALAN	54	5	2	2	1	1	2	4	RUSELL	1	NO	SELF	5	6
VIJAY	45	4	1	2	1	1	2	4	KRAIT	2	ORAL DRUGS	SELF	16	20
pounammal	60	6	2	1	1	3	1	2	NOT KNOWN	2	NO	PHYSICIAN	6	9
THIRUPAL	56	5	1	1	1	3	1	4	RUSELL	4	NO	SELF	11	12
MUNUSWAMY	57	5	1	1	1	1	2	2	RUSELL	2	NO	SELF	6	8
MUNIAPPAN	40	4	1	1	1	1	1	4	NOT KNOWN	3	NO	SELF	11	18
THANSAIMARY	38	3	2	2	2	2	2	4	NOT KNOWN	2	NO	SELF	1	24
JAYA	30	3	2	2	3	3	3	4	NOT KNOWN	2	NO	SELF	6	7
ULAGANATHAN	50	5	1	1	1	1	1	1	SAW SCALLED	3	NO	SELF	3	4
rajesh	28	2	1	1	3	1	3	4	RUSELL	3	NO	SELF	2	8
VAJRAVEL	36	3	1	1	3	1	3	4	NOT KNOWN	3	NO	SELF	3	8
NEDUCHEZIAN	44	4	1	1	1	1	1	4	NOT KNOWN	3	NO	SELF	5	9

completed	time of completion	TIME OF COMPLETION	asv /renal failure	dose	edema	lymph node	subconj	FLANG MARKS	spon. bleeding	HYPOTENSION	breathlessnees	neurological	uremia	oliguria
NO	26	2	NOT	8	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
NO	30	2	NOT	13	YES	YES	YES	YES	NO	NO	YES	NO	YES	YES
YES	22	1	YES	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	21	1	YES	18	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	23	1	YES	18	YES	YES	YES	NO	NO	NO	YES	NO	YES	YES
YES	15	1	YES	23	YES	NO	NO	NO	NO	NO	YES	YES	NO	NO
NO	30	2	NOT	0	YES	YES	YES	YES	NO	NO	NO	NO	NO	YES
YES	20	1	NOT	13	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	26	2	NOT	8	YES	NO	YES	YES	YES	HYPOTENSION	NO	NO	YES	ANURIC
YES	20	1	NOT	23	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO
YES	20	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	24	1	YES	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	34	2	NOT	5	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO
NO	NO	NO	NOT	13	YES	YES	YES	YES	YES	NO	YES	YES	YES	YES
YES	38	2	NOT	13	YES	YES	YES	NO	NO	NO	NO	NO	YES	NO
NO	28	2	NOT	13	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	24	1	YES	18	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	15	1	YES	23	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
NO	54	3	NOT	23	YES	YES	NO	NO	NO	NO	YES	YES	YES	YES
YES	17	1	YES	23	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO

YES	21	1	NOT	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	14	1	NOT	23	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO
YES	19	1	YES	18	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	20	1	NOT	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	30	2	YES	13	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO
NO	37	2	NOT	13	YES	YES	NO	NO	NO	NO	YES	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	24	1	YES	13	NO	NO	NO	NO	NO	NO	NO	NO	NO	OLIGURIC
NO	28	2	NOT	13	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	43	2	NOT	23	YES	YES	YES	YES	YES	NO	YES	NO	YES	ANURIC
YES	22	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	32	2	NOT	13	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	21	1	NOT	13	YES	YES	NO	YES	NO	NO	NO	NO	NO	YES
YES	25	2	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	34	2	NOT	5	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO
YES	26	2	NOT	13	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO
YES	22	1	NOT	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	24	1	YES	18	MILD	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	41	2	NOT	18	NO	NO	NO	NO	NO	NO	YES	NO	NO	OLIGURIC
YES	22	1	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	27	2	NOT	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
NO	30	2	NOT	13	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
NO	28	2	NOT	13	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO

YES	20	1	YES	18	YES	YES	NO	NO	NO	NO	NO	YES	NO	
YES	22	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	47	2	NOT	5	YES	YES	NO	NO	NO	NO	YES	YES	NO	NO
NO	26	2	NOT	8	MILD	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	24	1	YES	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	23	1	YES	18	YES	YES	NO	NO	NO	NO	YES	NO	YES	YES
NO	30	2	NOT	0	YES	YES	YES	YES	NO	NO	NO	NO	NO	YES
YES	16	1	YES	23	YES	NO	NO	NO	NO	NO	YES	YES	NO	NO
YES	14	1	YES	23	YES	NO	NO	NO	NO	NO	YES	YES	NO	NO
NO	19	1	NOT	18	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO
YES	40	2	NOT	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
NO	26	2	NOT	8	MILD	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	23	1	YES	18	YES	YES	NO	YES	NO	NO	YES	YES	YES	YES
YES	22	1	NOT	18	YES	YES	YES	NO	NO	NO	YES	NO	NO	YES
YES	34	2	NOT	5	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO
YES	24	1	YES	13	NO	NO	NO	NO	NO	NO	NO	NO	NO	OLIGURIC
SELF	42	2	NOT	8	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
NO	40	2	NOT	13	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO
YES	23	1	YES	18	YES	YES	NO	NO	NO	NO	YES	NO	YES	YES
YES	36	2	NOT	18	NO	NO	YES	YES	YES	NO	YES	NO	YES	ANURIC
NO	36	2	NOT	8	YES	NO	NO	NO	NO	NO	YES	NO	NO	YES
YES	23	1	YES	18	YES	YES	NO	YES	NO	YES	NO	NO	YES	YES
NO	38	2	NOT	13	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO

YES	30	2	YES	18	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	32	2	NOT	18	YES	NO	YES	NO	NO	NO	NO	NO	NO	YES
NO	28	2	NOT	13	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
NO	36	2	NOT	8	YES	NO	NO	NO	NO	NO	YES	NO	NO	YES
NO	19	1	NOT	18	YES	YES	NO	YES	NO	NO	NO	NO	NO	YES
YES	26	2	NOT	13	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO
YES	22	1	YES	18	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO
YES	26	2	NOT	13	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO
YES	33	2	NOT	13	YES	YES	NO	YES	NO	NO	NO	YES	NO	NO
YES	34	2	NOT	13	YES	YES	NO	YES	NO	NO	NO	YES	NO	NO

anuria	presentation	URINE	RBC	anemia	TCP	PT	aPTT	WBCT	WBCT NORMAL BY	AGMENTED RBC	1ST CREATININE	AKIN	SR.CR AT INITIATION	PEAK	BUN AT RESENTATION
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	20	2	NO	1.4	3	3.4	3.4	78
NO	LR	NO	NO	NO	NO	PROLONGED	PROLONGED	23	2	NO	3.1	3	4.5	6.7	122
NO	H	NO	NO	NO	NO	PROLONGED	PROLONGED	18	2	NO	1.1	2	2.1	2.1	88
NO	LH	NO	NO	YES	NO	PROLONGED	PROLONGED	21	2	NO	1	2	1.6	1.9	86
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	39	4	NO	3.6	3	3.6	6.1	102
NO	LN	NO	NO	NO	NO	NO	NO	NO	1	NO	1.2	3	3.1	3.7	97
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	14	2	NO	3.4	3	3.4	5.7	67
NO	LH	NO	NO	NO	NO	NO	NO	20	2	NO	0.9	2	1.8	1.8	72
ANURIC	LHR	NO	YES	YES	YES	PROLONGED	PROLONGED	22	2	YES	9.3	3	9.3	9.3	163
NO	N	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.1	4.1	77
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	0.9	2	1.9	2	102
NO	LR	NO	NO	NO	NO	PROLONGED	PROLONGED	18	2	NO	2.3	3	3.4	4.4	102
NO	LHR	TRACE	YES	YES	NO	PROLONGED	PROLONGED	26	3	NO	2.3	3	3.9	7.1	88
ANURIC	LHNR	NO	YES	YES	YES	PROLONGED	PROLONGED	PRONGED	4	YES	6.7	3	6.7	6.7	99
NO	LHR	NO	YES	YES	NO	PROLONGED	PROLONGED	36	3	NO	2.1	3	3.9	6.6	98
NO	LH	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.2	5.6	66
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	1	1.5	1.9	78
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	0.7	2	1.5	1.5	71
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	PRONGED	4	NO	0.9	3	3.1	4.6	103
ANURIC	LHNR	NO	NO	NO	NO	PROLONGED	PROLONGED	PRONGED	4	YES	3.4	3		7.9	88
NO	N	NO	NO	NO	NO	NO	NO	NO	1	NO	1.2	3	3.2	4.1	77

NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	PRONGED	4	YES	1.2	3	2.9	6.1	56
NO	N	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	2.1	3.9	56
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	0.6	3	2	2.4	132
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	2	2.2	2.6	121
ANURIC	LHR	NO	YES	YES	YES	PROLONGED	PROLONGED	40	4	YES	1.3	3	3.4	5.6	88
NO	L	NO	NO	NO	NO	NO	NO	18	2	NO	1.9	2	1.9	5.4	66
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	HR	NO	NO	NO	NO	PROLONGED	PROLONGED	18	2	NO	2.3	3	3.4	4.4	102
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.2	5.6	66
NO	LR	NO	NO	NO	NO	NO	NO	NO	1	NO	2.9	3	3	6.5	84
ANURIC	HR	NO	YES	YES	YES	PROLONGED	PROLONGED	36	3	YES	2.8	3	5.9	8	98
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	2	2.3	3	132
NO	LHR	NO	NO	YES	NO	PROLONGED	PROLONGED	34	3	NO	1	3	3.7	3.9	109
NO	L	NO	NO	NO	NO	NO	NO	25	3	NO	1.1	2	1.9	2.5	89
NO	LHR	SUBNEPH.	YES	YES	YES	PROLONGED	PROLONGED	28	3	YES	3.4	3	3.4	6.9	74
NO	LH	SUBNEPH.	YES	YES	YES	PROLONGED	PROLONGED	28	3	YES	1.5	3	4.2	7.1	66
NO	L	NO	NO	NO	NO	NO	NO	22	2	NO	0.9	2	1.9	1.9	67
NO	L	NO	NO	NO	NO	NO	NO	22	2	NO	0.9	2	1.8	1.9	63
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.6	3	3.1	4.2	73
NO	HR	NO	NO	YES	NO	PROLONGED	PROLONGED	34	3	NO	3.9	3	3.9	5.1	98
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	18	2	NO	1.1	1	1.6	4.1	68
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	0.8	2	2.1	2.65	76
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	26	3	NO	1.9	3	3.6	4.5	67
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.2	5.6	66

NO	LHN	NO	NO	YES	NO	PROLONGED	PROLONGED	18	2	NO	1.1	2	2.3	3.7	88
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	LN	NO	NO	NO	NO	NO	NO	NO	1	NO	1.2	3	3.4	4.2	78
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	26	3	NO	1.1	3	3.4	3.4	79
NO	L	NO	NO	NO	NO	NO	NO	24	2	NO	1.1	1	1.6	1.7	67
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	39	4	NO	3.6	3	3.6	6.1	102
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	14	2	NO	3.4	3	3.4	5.7	67
NO	LN	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.1	4.1	76
NO	LN	NO	NO	NO	NO	NO	NO	NO	1	NO	0.9	3	4.1	6.4	139
NO	N	NO	NO	YES	NO	NO	NO	19	2	NO	1.1	1	1.6	2.4	101
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	38	4	NO	1.1	3	3.6	5.2	94
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	3	3.5	4.9	78
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.5	3.6	96
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	24	2	NO	0.9	3	2.9	3.9	96
NO	LHNR	NO	NO	NO	NO	PROLONGED	PROLONGED	39	4	NO	3.6	3	3.6	6.1	102
NO	LHR	SUBNEPH.	YES	YES	YES	PROLONGED	PROLONGED	20	2	NO	3.1	3	4.1	7.4	66
NO	LHR	TRACE	YES	YES	NO	PROLONGED	PROLONGED	26	3	NO	2.5	3	3.9	4.9	77
NO	HR	NO	NO	NO	NO	PROLONGED	PROLONGED	18	2	NO	2.3	3	3.4	4.4	102
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.3	2	2.9	4.8	69
NO	LR	NO	NO	NO	NO	NO	NO	NO	1	NO	2.7	3	5.9	8.9	102
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	39	4	NO	3.6	3	3.6	5.5	76
ANURIC	HR	SUBNEPH.	YES	YES	YES	PROLONGED	PROLONGED	18	2	YES	3.1	3	4.7	10	81
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	32	3	NO	2.4	3	4.5	5.1	83
NO	LHR	NO	NO	YES	NO	PROLONGED	PROLONGED	24	2	YES	5.7	3	5.7	5.7	89
NO	N	NO	NO	NO	NO	NO	NO	34	3	NO	0.7	2	1.5	1.8	81

NO	LR	NO	NO	NO	NO	NO	NO	18	2	NO	2.1	3	3.4	4.9	78
ANURIC	LHR	TRACE	YES	YES	YES	PROLONGED	PROLONGED	26	3	YES	3.1	3	3.9	9.2	94
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.1	3	3.2	5.9	66
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	34	3	NO	1.4	3	3.6	5.1	83
NO	LHR	TRACE	YES	YES	NO	PROLONGED	PROLONGED	34	3	NO		3		6	
NO	L	SUBNEPH.	NO	NO	NO	NO	NO	19	2	NO	1.3	3	4.2	6.9	66
NO	L	NO	NO	NO	NO	NO	NO	NO	1	NO	1.2	3	3	4.5	84
NO	LH	NO	NO	NO	NO	PROLONGED	PROLONGED	21	2	NO	5	3	5.3	5.3	99
NO	LHN	NO	NO	NO	NO	PROLONGED	PROLONGED	27	3	NO	4.6	3	4.6	4.6	66
NO	LHR	NO	NO	NO	NO	PROLONGED	PROLONGED	28	3	NO	5.4	3	6.9	6.9	112

K AT PTESENTATION	2ND	HYPERCAT ABOLIC	URINE MYO	BILURUBIN	PEAK	LDH	CPK PEAK	DIC WORK UP	RF TIME	rf TIME	DIALSIS	HD/P D	DIA. TIME	SR.CR	ASV	OUTPUT	INDICATION
4.5	3.9	NO	NA	N	NA	N	N	N	48	3	YES	HD	3RD	3.4	18	OLIGURIC	OLIGURIA AND ACIDOSIS
3.9	4.9	YES	NA	N	N	N	N	N	18	1	YES	HD	1ST	4.5	23	OLIGURIC	OLIGURIA AND ACIDOSIS
4.7	4.4	NO	NA	N	NA	NA	N	N	27	2	NO	NO	NA	NA	18	NA	NA
4.1	4	NO	NA	N	NA	NA	N	N	54	3	NO	NO	NA	NA	18	NA	NA
4.9	5.7	YES	N	N	NA	284	465	YES	22	1	YES	HD	2ND	3.6	23	OLIGURIC	OLIGURIA AND ACIDOSIS
5.2	4.9	NO	NA	N	NA	NA	640	NA	28	2	YES	HD	2ND	3.1	23	OLIGURIC	OLIGURIA AND RHABDOMYOLYSIS
4.5	5.6	YES	NA	N	NA	NA	322	N	14	1	YES	HD	1ST	3.4	13	OLIGURIC	OLIGURIC AND ACIDOSIS
4.5	4.4	NO	NA	N	NA	NA	N	N	36	2	NO	NA	NA	NA	18	NA	NA
4.4	5.4	YES	POSITIVE	4.7	8	534	1340	YES	24	2	YES	PD	2ND	9.3	23	ANURIC	ANURIA
4.4	4.2	NO	NA	N	NA	NA	N	N	36	2	YES	HD	2ND	3.4	23	OLIGURIC	ACIDOSIS
4.6	3.4	NO	NA	N	NA	NA	NA	N	25	2	NO	NA	NA	NA	18	OLIGURIC	NA
4.9	4.4	NO	NA	N	NA	NA	N	N	15	1	YES	HD	2ND	3.4	18	OLIGURIC	ACIDOSIS
4.4	5.7	YES	N	N	NA	NA	N	YES	14	1	YES	HD	1ST	3.9	5	OLIGURIC	ACIDOSIS
3.8	4.9	YES	POSITIVE	6.1	6.1	440	3400	YES	15	1	YES	HD	1ST	6.7	5	ANURIC	ANURIC
4.9	5.9	YES	NA	N	NA	NA	433	N	24	2	YES	HD	2ND	6.6	23	OLIGURIC	ACIDOSIS
3.4	4.3	NO	NA	N	NA	NA	N	N	20	1	YES	HD	2ND	3.2	18	OLIGURIC	OLIGURIA
3.4	3.9	NO	NA	N	NA	NA	NA	N	44	2	NO	NA	NA	NA	18	NA	NA
3.5	NA	NO	NA	N	NA	NA	NA	N	52	3	NO	NO	NA	NA	18	NA	NA

NA	NA	NO	NA	N	NA	NA	N	N	210	8	YES	PD	14TH	3.1	23	ANURIC	ANURIC
4.7	5.9	YES	POSITIVE	2.4	5.6	731	2389	YES	40	2	YES	HD	2ND	3.9	23	OLIGURIC	OLIGURIA AND ACIDOSIS
4.6	4.3	NO	NA	N	NA	NA	N	N	24	2	YES	HD	2ND	3.2	23	OLIGURIC	ACIDOSIS
3.4	5.1	YES	NA	N	NA	365	NA	YES	29	2	YES	HD	2ND	3.4	18	OLIGURIC	OLIGURIA
3.5	3.4	NO	NA	N	NA	NA	N	N	34	2	YES	HD	3RD	3.9	23	OLIGURIC	ACIDOSIS
4.1	NA		NA	N	NA	NA	NA	N	66	3	NO	NO	NA	NA	18	NA	NA
3.8	NA		NA	N	NA	NA	NA	N	16	1	NO	NO	NA	NA	18	NA	NA
5.1	5.4	YES	NA	3.2	3.2	334	340	YES	16	1	YES	HD	1ST	3.4	23	OLIGURIC	OLIGRIC
4.3	5.1	YES	POSITIVE	N	NA	423	894	N	18	1	YES	HD	2ND	5.1	18	OLIGURIC	OLIGURIA AND RHABDOMYOLYSIS
3.4	4.9	NN	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
4.9	4.4	NO	NA	N	NA	NA	N	N	15	1	YES	HD	2ND	3.4	18	OLIGURIC	ACIDOSIS
3.4	4.3	NO	NA	N	NA	NA	NA	N	20	1	YES	HD	2ND	3.2	18	OLIGURIC	OLIGURIA
4.3	5.1	YES	YES	N	NA	406	2089	N	32	2	YES	HD	2ND	3	18	0LIGURIA	RHABDOMYOLYSIS
4.2	5.6	YES	POSITIVE	2.3	2.9	237	673	YES	22	1	YES	HD	2ND	4.1	18	ANURIC	OLIGURIA AND ACIDOSIS
3.4	4.9	NO	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
3.4	3.6	NO	NA	N	NA	NA	NA	N	22	1	NO	NO	NA	NA	18	NA	NA
3.4	3.9	NO	NA	N	NA	NA	NA	N	30	2	YES	HD	3RD	3.9	18	OLIGURIC	OLIGURIA
3.5	4.1	NO	NA	N	NA	NA	N	N	54	3	NO	NA	NA	NA	18	NA	NA
4.4	5.7	YES	NA	2.4	5.6	556	340	YES	24	1	YES	HD	1ST	3.4	18	OLIGURIC	ACIDOSIS
3.4	5.1	YES	NA	3.1	4.2	766	432	YES	20	1	YES	HD	2ND	7.6	23	OLIGURIC	OLIGURIA
3.4	3.8	NO	NA	N	NA	NA	N	N	56	3	NO	NO	NA	NA	18	NA	NA
3.6	3.9	NO	NA	N	NA	NA	N	N	47	2	NO	NO	NA	NA	18	NA	NA
5.2	4.5	NO	NA	N	NA	367	748	N	24	2	YES	HD	3RD	3.1	18	NONOLIGURIC	HYPERKALEMIA
4.1	5.1	YES	NA	N	NA	NA	N	N	26	2	YES	HD	2ND	3.9	18	OLIGURIC	ACIDOSIS

3.7	4.9	YES	NA	N	NA	NA	N	N	27	2	YES	HD	2nd	4.1	18	OLIGURIC	OLIGURIA AND ACIDOSIS
4.1	3.4	NO	NA	N	NA	NA	NA	N	37	2	NO	NA	NA	NA	18	NA	NA
3.4	4.9	NO	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
3.4	4.6	YES	NA	N	NA	NA	N	N	28	2	YES	HD	2ND	3.6	18	OLIGURIC	OLIGURIA
3.4	4.3	NO	NA	N	NA	NA	NA	N	20	1	YES	HD	2ND	3.2	18	OLIGURIC	OLIGURIA
3.7	4.4	NO	NA	N	NA	NA	N	N	34	2	YES	HD	3RD	3.7	18	OLIGURIC	OLIGURIA
3.4	4.9	NO	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
3.4	4.9	YES	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
43	4.4	NO	NA	N	NA	NA	N	N	36	2	YES	HD	2ND	3.4	23	OLIGURIC	OLIGURIA AND ACIDOSIS
3.5	3.4	NO	NA	N	NA	NA	N	N	23	1	YES	HD	3RD	3.4	18	OLIGURIC	OLIGURIA
3.6	3.4	NO	NA	N	NA	NA	N	N	70	3	NO	NO	NA	NA	18	NA	NA
4.9	5.7	NO	NA	N	NA	NA	N	N	22	1	YES	HD	2ND	3.6	23	OLIGURIC	OLIGURIA AND ACIDOSIS
5.1	5.6	YES	NA	N	NA	NA	322	N	14	1	YES	HD	1ST	3.4	13	OLIGURIC	OLIGURIC AND ACIDOSIS
4.9	3.2	NO	NA	N	NA	NA	360	N	28	2	YES	HD	2ND	3.1	23	OLIGURIC	OLIGRIC
5.3	5.1	NO	NA	N	NA	NA	NA	N	412	14	YES	PD	16TH	4.1	23	ANURIC	ANURIC
3.5	NA	NO	NA	N	NA	NA	NA	N	56	2	NO	NO	NA	NA	18	NA	NA
4.7	4.4	NO	NA	N	NA	NA	N	N	38	2	YES	HD	3RD	5.4	18	OLIGURIC	ACIDOSIS
3.4	4.9	NO	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
3.4	4.6	NO	NA	N	NA	NA	NA	N	44	2	YES	HD	2ND	4.1	18	OLIGURIC	ACIDOSIS
3.8	3.1	NO	NA	N	NA	NA	N	N	22	1	YES	HD	3RD	2.9	18	OLIGURIC	OLIGURIA
4.5	5.7	YES	NA	N	NA	NA	N	N	22	1	YES	HD	2ND	3.6	23	OLIGURIC	OLIGURIA AND ACIDOSIS
3.6	5.1	YES	NA	N	NA	NA	N	N	14	1	YES	HD	2ND	3.9	23	OLIGURIC	ANURIC

5.4	5.7	NO	NA	N	NA	344	N	YES	14	1	YES	HD	1ST	3.9	5	OLIGURIC	ACIDOSIS
4.9	4.4	NO	NA	N	NA	NA	N	N	15	1	YES	HD	2ND	3.4	18	OLIGURIC	ACIDOSIS
3.5	4.6	NO	NA	N	NA	NA	NA	N	48	3	YES	HD	3RD	3.2	18	OLIGURIC	ACIDOSIS
5.1	5.9	YES	POSITIVE	N	NA	432	986	N	36	2	YES	HD	2ND	5.9	18	OLIGURIC	RHABDOMYOLYSIS
4.9	5.7	NO	NA	N	NA	NA	N	N	22	1	YES	HD	2ND	3.6	18	OLIGURIC	OLIGURIA AND ACIDOSIS
4.1	5.6	YES	POSITIVE	3.2	4.4	451	874	YES	22	1	YES	HD	2ND	4.1	18	ANURIC	OLIGURIA AND ACIDOSIS
3.4	4.7	NO	NA	N	NA	N	N	N	33	2	YES	HD	3RD	4.5	18	OLIGURIC	OLIGURIA AND ACIDOSIS
3.4	3.6	NO	NA	N	NA	NA	NA	YES	72	4	YES	HD	3RD	5.9	18	OLIGURIC	OLIGURIC AND ACIDOSIS
3.4	3.3	NO	NA	N	NA	NA	N	N	34	2	NO	NO	NA	NA	18	NA	NA
3.6	4.5	NO	NA	N	NA	NA	NA	N	22	1	YES	HD	2ND	3.4	18	NONOLIGURIC	ACIDOSIS
4.1	5.2	YES	NA	2.6	3.2	379	1095	YES	18	1	YES	HD	2ND	5.1	23	OLIGURIC	OLIGURIA
3.4	4.3	NO	NA	N	NA	NA	NA	N	20	1	YES	HD	2ND	3.2	18	OLIGURIC	OLIGURIA
3.4	4.7	YES	NA	N	NA	N	N	N	34	2	YES	HD	3RD	3.6	18	OLIGURIC	OLIGURIA AND ACIDOSIS
		YES	NA					NO			YES	HD			18		
4.1	4.4	NO	NA	N	NA	NA	N	N	16	1	YES	HD	2ND	4.2	18	OLIGURIC	OLIGURIA
4.3	3.9	NO	NA	N	NA	NA	NA	N	32	2	YES	HD	2ND	4.4	18	0LIGURIA	RHABDOMYOLYSIS
3.9	5.2	NO	NO	N	NA	NA	N	N	24	2	YES	HD	2ND	5.3	23	OLIGURIC	OLIGRIA AND ACIDOSIS
34	4.5	NO	NO	N	NA	NA	N	N	22	1	YES	HD	2ND	4.6	23	OLIGURIC	ACIDOSIS
3.4	4.5	NO	NO	N	NA	NA	N	N	23	1	YES	HD	2ND	4.5	23	OLIGURIC	ACIDOSIS

TOTAL	DURATION	IMPR.OUTPUT	BASELINE	BIOPSY	OUTCOME	CAUSE OF DEATH	TIME OF DEATH	VENTILATOR	POST CARDIAC ARREST	MEDICAL	DIS .CR	FOLLOW UP EGFR	LATEST
4 SESSIONS	8DAYS	9 DAYS	13	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1	N	NA
3	4	NA	NA	NOTDONE	EXPIRED	SEPSIS	5TH	YES	YES	SEPSIS	NA	EXPIRED	EXPIRED
NA	NA	NA	6	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
NA	NA	NA	6	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	0.9	N	NA
8	17	16	28	ATN	RECOVERED	NA	NA	NO	NO	NA	1.8	82	82
4	8	5	8	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
5	9	5	17	NOTDONE	PARTIAL RECOVERY	NA	NA	NO	NO	NA	1.7	78	72
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
PD	2	NA	NA	NOTDONE	EXPIRED	DIC SEPSIS	3 DAYS	YES	NO	DIC SEPSIS	NA	EXPIRED	EXPIRED
4	9	7	16	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	0.9	N	NA
3	6	4	9	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.3	N	NA
8	19	OLIGURIC	NA	CAN	PARTIAL RECOVERY	NA	NA	NO	NO	NA	2.1	74	86
1	1	NA	NO	NOTDONE	EXPIRED	DIC	1 DAY	YES	YES	DIC SEPSIS	EXPIRED	EXPIRED	EXPIRED
9	17	14	NA	ATN	PARTIAL RECOVERY	NA	NA	NO	NO	NA	2.9	25	25
5	8	6	13	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
PD	2	NA	NA	NOTDONE	EXPIRED	VAP	21DAYS	YES	YES	SEPSIS		EXPIRED	EXPIRED

2	2	NA	NO	NOTDONE	EXPIRED	DIC AND RHABDOM YOLYSIS		NO	NO	NA	EXPIRED	EXPIRED	EXPIRED
6	11	8	15	NOTDONE	NA	NA	NA	NO	NO	NA	1.1	N	NA
11	21	15	33	ATN	RECOVERED	NA	NA	NA	NA	NA	1.2	N	NA
4		7	11	NOTDONE	NA	NA	NA	NO	NO	NA	0.9	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.2	N	NA
5	8	NA	NA	NOTDONE	EXPIRED	SEPSIS	9TH	YES	YES	DIC SEPSIS	NA	EXPIRED	EXPIRED
5	11	8	17	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
4	6	4	9	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.3	N	NA
4	8	6	13	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
11	26	21	34	CAN	PARTIAL RECOVERY	NA	NA	NA	NA	NA	2.1	68	60
9	21	16	NO	ATN	PARTIAL	NA	NA	NA	NA	NA	1.8	2.1	76
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	0.9	N	NA
4	6	5	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
11	26	OLIGURIC	NO	CAN	PARTIAL RECOVERY	NA	NA	NO	NO	NA	2.6	56	45
EXPIRED	EXPIRED	EXPIRED	EXPIRED	TMA	EXPIRED	SEPSIS	25	YES	NO	SEPSIS	EXPIRED	EXPIRED	EXPIRED
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA

3	6	NONOLIGURIC	9	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.2	N	N
8	16	11	19	ATIN	PARTIAL RECOVERY	NA	NA	NA	NA	NA	1.6	56	56
4	6	5	9	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED		NA	NO	NO	NA	0.9	N	NA
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
4	6	7	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
5	8	6	13	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1	N	NA
4	7	8	12	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	0.9	N	NA
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
ON MHD	ON MHD	OLIGURIC	MHD	ATN	NO RECOVERY	NA	NA	NA	NA	NA	4.3	ON MHD	24
3 SESSIONS	5DAYS	4DAYS	9	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
8	19	18	28	ATN	RECOVERED	NA	NA	NA	NA	NA	1.8	82	82
5	9	5	NO	NOTDONE	PARTIAL RECOVRY	NA	NA	NO	NO	NA	1.7	82	72
3	5	4	9	NOTDONE	NA	NA	NA	NA	NA	NA	1	N	NA
PD	2	NA	NA	NOTDONE	EXPIRED	VAP	9 DAYS	YES	YES	NA	NA	EXPIRED	EXPIRED
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
4	7	8	10	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
4	7	6	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.1	N	NA
5	9	7	13	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	0.9	N	NA
4 SESSIONS	8DAYS	7TH	11	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.3	N	NA
10	17	14	NO	ATN	PARTIAL RECOVERY	NA	NA	NA	NA	NA	1.9	72	EXPIRED

6	11	8	21	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.3	N	NA
4	8	7	12	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
4	6	4	9	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.3	N	NA
5	8	7	15	NOTDONE	RECOVERED	NA	NA	RECOVERED	NA	NA	1.4	N	NA
8	17	19	26	ATN	PARTIAL	NA	NA	NA	NA	NA	1.4	78	78
5	9	10	16	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.1	N	NA
12	24	15	NO	CAN DONE OUTSIDE	PARTIAL RECOVERY	NA	NA	NA	NA	NA	2.4	2.1	66
5	9	9 DAYS	14 DAYS	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.4	N	NA
2	DEATH	DEATH	DEATH	NOTDONE	EXPIRED	DIC	5	YES	YES	SEPSIS	DEATH	N	NA
NA	NA	NA	NA	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	1.4	NA	NA
11	22	NO	NO	ATIN	EXPIRED	CRBSI	22	NO	NO	NO	EXPIRED	EXPIRED	EXPIRED
12	26	OLIGURIC	NO	TMA	PARTIAL	NA	NA	NO	NA	NA	5.4	25	ON MHD
6	11	6	16	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.2	N	NA
7	16	13	21	NOTDONE	RECOVERED	NA	NA	NO	NO	NA	1.4	N	NA
				CAN	PARTIAL RECOVERY	NA	NA	NA	NA	NA	5.6	ON MHD	MHD
9	21	16	32	ATIN	NA	NA	NA	NA	NA	NA	1.1	1.1	NA
6	11	8	11	NOTDONE	RECOVERED	NA	NA	NA	NA	NA	N	NA	NA
5	7	8	13	NOTDONE	NA	NA	NA	NA	NA	NA	1.3	1.1	NA
4	15	17	21	ATN	RECOVERED	NA	NA	NA	NA	NA	1.1	1.1	NA
7	17	15	21	ATN	RECOVERED	NA	NA	NA	NA	NA	1.1	1.1	NA

NAME	AGE	AGE	SEX	EDUCATION	SOCIO ECONOMIC	AMOUNT	ALCOHOL	KNOWN	TIME LAG TO DIAGNOSIS	IST SPOT	TIME CONSUMED	TREATMENT AT IST SPOT	REFERAL TO DIALYS	TOTAL LAG	LAG	PREUMPTIVE HD	PRESENTATION	HB
MUTHU	45	4	2	1	1	4	ALCOHOLIC	NO	4	1	4	EARLY DECONTAMINATION	INDIRECT	48	3	NO	COMATOSE, SEIZURES	11.2
MANIKANDAN	32	3	2	1	1	3	ALCOHOLIC	YES	4	1	2	EARLY DECONTAMINATION	DIRECT	9	1	YES	SEIZURE	10.4
KATHIRVEL	28	2	2	1	1	4	ALCOHOLIC	YES	1	2	8	EARLY DECONTAMINATION	DIRECT	11	1	YES	SEIZURE	11.9
SATHYA	28	2	1	2	2	3	NON	NOTKNOWN	6	1	2	EARLY DECONTAMINATION	DIRECT	28	2	NO	COMATOSE, SEIZURES	7.1
MANICKAM	30	3	2	1	2	3	NON	YES	1	1	2	EARLY DECONTAMINATION	INDIRECT	6	1	YES	GI	10
MARAGATHAM	26	2	1	3	1	2	NONALCOHOLIC	KNOWN	1	2	44	DECONT,AND SUPPORTIVE	DIRECT	46	3	NO	RENAL	8.1
KALAIARASI	39	3	1	2	1	4	NON	YES	1	2	4	EARLY DECONTAMINATION	DIRECT	10	1	YES	GI	9.4
DHAYALAN	46	4	2	3	2	4	ALCOHOLIC	YES	2	1	4	EARLY DECONTAMINATION	DIRECT	8	1	YES	GI	9.3
KUMAR	36	3	2	1	2	1	NONALCOHOLIC	NOTKNOWN	1	2	36	DECONTAMINATION AND SUPPORTIVE	DIRECT	40	2	NO	GI,RENAL	10.3
KESAVAN	50	5	2	1	1	2	NONALCOHOLIC	KNOWN	2	3	1	EARLY DECONTAMINATION	DIRECT	8	1	YES	GI	9.3
MUTHU	25	2	2	2	2	3	ALCOHOLIC	NOTKNOWN	2	2	4	EARLY DECONTAMINATION	DIRECT	24	1	NO	GI	10.1
THIRMALAI	40	4	2	1	2	2	NONALCOHOLIC	NOTKNOWN	3	2	12	DECONT,AND SUPPORTIVE	DIRECT	48	3	NO	GI,RENAL	8.9
BALAJI	18	1	2	1	1	2	ALCOHOLIC	N0TKNOWN	6	1	2	EARLY DECONTAMINATION	DIRECT	28	2	NO	COMATOSE, SEIZURES	9.9
VELU	25	2	2	1	1	2	ALCOHOLIC	KNOWN	2	1	2	EARLY DECONTAMINATION	DIRECT	9	1	YES	SEIZURE	10.4

MURALI	21	2	2	3	2	2	ALCOHOLIC	NOTKNOWN	4	2	48	DECONT,AND SUPPORTIVE	DIRECT	56	3	NO	RENAL	8.4
ASHOK	25	2	2	1	1	3	ALCOHOLIC	NOTKNOWN	2	2	4	EARLY DECONTAMINATION	DIRECT	30	2	NO	GI	11.4
VIMALA	29	2	1	2	1	2	NONALCOHOLIC	NOTKNOWN	4	2	36	DECONT,AND SUPPORTIVE	DIRECT	46	3	NO	RENAL	7.9
NAGARA	23	2	2	1	2	2	NONALCOHOLIC	KNOWN	1	2	72	DECONT,AND SUPPORTIVE	DIRECT	76	4	NO	RENAL	11.3
RAJEEV GANDHI	23	2	2	3	1	2	NONALCOHOLIC	KNOWN	1	2	79	DECONT,AND SUPPORTIVE	DIRECT	85	4	NO	RENAL	11
ARULRAJ	20	2	2	3	1	2	ALCOHOLIC	NOTKNOWN	4	2	88	DECONT,AND SUPPORTIVE	DIRECT	96	4	NO	RENAL	10.1
VARADARAJAN	36	3	2	3	1	2	ALCOHOLIC	NOTKNOWN	4	3	12	DECONTAMINATION AND SUPPORTIVE	DIRECT	14	1	YES	GI	11
VIJAYAKUMR	22	2	2	1	1	3	ALCOHOLIC	YES	1	2	44	DECONT,AND SUPPORTIVE	DIRECT	46	3	NO	RENAL	8.1
AMULRAJ	20	2	2	1	1	3	NONALCOHOLIC	YES	1	2	4	EARLY DECONTAMINATION	DIRECT	10	1	YES	GI	9.4
RAMESH	30	3	2	1	1	4	ALCOHOLIC	YES	2	1	4	EARLY DECONTAMINATION	DIRECT	8	1	YES	GI	9.3
JAMUNA	17	1	1	1	1	4	NONALCOHOLIC	YES	1	2	36	DECONTAMINATION AND SUPPORTIVE	DIRECT	40	2	NO	GI,RENAL	10.3

PULMONARY	SPO2/ PAO2 P	R	ARDS	PULSE	BP	VENTILATOR	CARDIAC ARREST	HEPATIC	CNS	O.P	C.P	B.P	TIME OF RF
YES	<200	<200	YES	STERIOD	HYPOTENSION	YES	YES	NO	YES	ANURIC	4.9	96	48 hrs
NO	<500	<500	NO	NO	N	NO	NO	NO	YES	NONOLIGURIC	1.1	33	72
YES	<200	<200	YES	STERIOD	N	YES	NO	NO	YES	NONOLIGURIC	1.1	49	54
NO	<500	<500	NO	NO	N	NO	NO	NO	YES	OLIGURIC	1.6	45	28
YES	<500	<200	YES	NO	N	YES	NO	NO	NO	NONOLIGURIC	0.9	39	74
YES	<500	<500	NO	NO	N	NO	NO	MILD	NO	OLIGURIC	2.3	88	AT PRESEN
YES	<500	<200	YES	STERIOD	N	YES	NO	YES	NO	NONOLIGURIC	1	39	18
YES	<500	<200	YES	STERIOD	N	YES	NO	YES	NO	NONOLIGURIC	1.1	64	34
YES	>500	>500	NO	NO	N	NO	NO	NO	NO	NONOLIGURIC	2.9	74	38
YES	<500	<200	YES	NO	HYPOTENSION	YES	NO	YES	YES	OLIGURIC	1.3	66	36
YES	<200	<200	YES	NO	N	YES	NO	YES	NO	OLIGURIC	2.2	66	AT PRESEN
YES	<500	<500	NO	NO	N	NO	NO	MILD	NO	OLIGURIC	1.4	77	48
NO	<500	<500	NO	NO	N	NO	NO	NO	YES	OLIGURIC	1.8	66	AT PRESEN
NO	<500	<500	NO	NO	N	NO	NO	NO	YES	NONOLIGURIC	1.1	33	72
YES	<500	<500	NO	NO	N	NO	NO	NO	NO	OLIGURIC	2.4	68	AT PRESEN
YES	<200	<200	YES	NO	HYPOTENSION	YES	YES	YES	NO	OLIGURIC	1.9	59	AT PRESEN
YES	<500	<500	NO	NO	N	NO	NO	NO	NO	NONOLIGURIC	2.1	68	AT PRESEN
YES	<500	<200	YES	NO	N	YES	NO	MILD	NO	OLIGURIC	3.2	88	AT PRESEN
YES	<500	<200	YES	STERIOD	N	YES	NO	NO	NO	OLIGURIC	3.3	112	AT PRESEN
YES	<500	<200	YES	STERIOD	N	NO	NO	NO	NO	OLIGURIC	21	134	AT PRESEN
YES	<200	<20	YES	NO	HYPOTENSION	YES	YES	YES	YES	ANURIC	1.1	66	16
YES	<500	<500	NO	NO	N	NO	NO	MILD	NO	OLIGURIC	2.3	88	AT PRESEN
YES	<500	<200	YES	STERIOD	N	YES	NO	YES	NO	NONOLIGURIC	1	39	18
YES	<500	<200	YES	STERIOD	N	YES	NO	YES	NO	NONOLIGURIC	1.1	64	34
YES	>500	>500	NO	NO	N	NO	NO	NO	NO	NONOLIGURIC	2.9	74	38

O.R	C.R	B.R	INDICATION FOR HD	TOTAL SESSION	DURATION	OUTCOME	OUTPUT IMPROVED	C.B	CAUSE OF DEATH	TIME	C.D	C.F	LONG TERM
NA	NA	NA	ANURIC	PD	NA	DEATH	DEATH	NA	ARDS	3	DEATH	DEATH	DEATH
NONOLIGURI	2.1	66	NA	4	6	RECOVERED	NONOLIGURIC	7	NA	NA	0.9	NA	NA
OLIGURIC	3.3	98	ACIDOSIS	4	6	DEATH	DEATH	NA	ARDS	7	DEATH	DEATH	DEATH
OLIGURIC	NA	NA	ACIDOSIS	6	9	EXPIRED	8	13	NA	NA	1.1	NA	NA
NONOLIGURI	1.7	69	NA	3	5	RECOVERED	NONOLIGUIC	8	NA	NA	0.8	NA	NA
NONOLIGURIC	NA	NA	NA	3	5	RECOVERED	4	7	NA	NA	1	NA	NA
ANURIC	2.4	85	ANURIC	2	2	DEATH	DEATH	NA	ARDS	3	DEATH	DEATH	DEATH
OLIGURIC	3.1	97	ACIDOSIS	1	7	DEATH	NA	NA	ARDS	9	DEATH	DEATH	DEATH
NONOLIGURI	2.9	74	ACIDOSIS	2	2	RECOVERED	NONOLIGURIC	6	NA	NA	1.1	NA	NA
OLIGURIC	3.2	132	ACIDOSIS	PD	NA	DEATH	DEATH	NA	ARDS,CARDIOGENIC SHOCK	3	DEATH	DEATH	DEATH
OLIGURIC	NA	NA	ACIDOSIS	5	8	DEATH	DEATH	NA	ARDS	9	DEATH	DEATH	DEATH
OLIGURIC	2.7	88	ACIDOSIS	5	10	RECOVERED	7	11	NA	NA	1	N	NA
OLIGURIC	NA	NA	ACIDOSIS	6	9	RECOVERED	6	11	NA	NA	1	NA	NA
NONOLIGURI	2.1	66	NA	4	6	RECOVERED	NONOLIGURIC	7	NA	NA	0.9	NA	NA
OLIGURIC	NA	NA	NA	4	6	RECOVERED	4	6	NA	NA	1.2	NA	NA

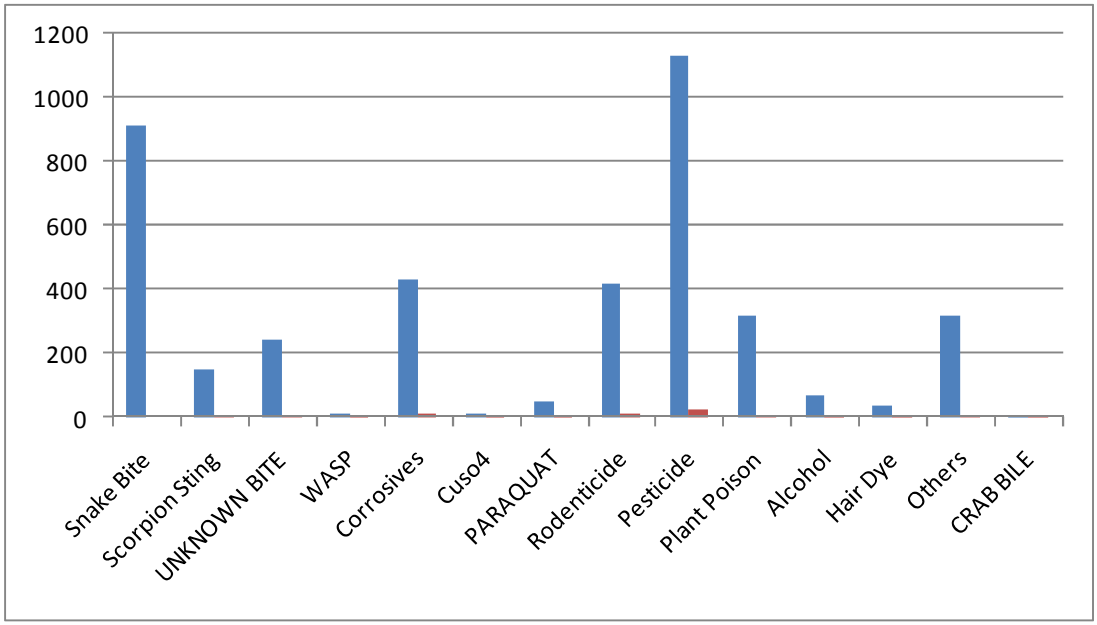
ANURIC	NA	NA	ANURIA	PD	NA	DEATH	DEATH	NA	CARDIOGENIC SHOCK,ARDS	3	DEATH	DEATH	DEATH
NONOLIGURIC	NA	NA	NA	2	4	RECOVERED		6	NA	NA	0.9	NA	NA
OLIGURIC	NA	NA	NA	6	9	RECOVERED	8	11	NA	NA	1.2	NA	NA
OLIGURIC	NA	NA	NA	4	6	RECOVERED	9	11	NA	NA	1.1	NA	NA
ANURIC	NA	NA	ACIDOSIS	2	3	DEATH	DEATH	NA	ARDS	5	DEATH	DEATH	DEATH
ANURIC	1.9	99	ACIDOSIS	2	4	DEATH	DEATH	NA	ARDS	5	DEATH	DEATH	DEATH
NONOLIGURIC	NA	NA	NA	3	5	DEATH	DEATH	NA	ARDS	3	1	NA	NA
ANURIC	2.4	85	ANURIC	2	2	DEATH	DEATH	NA	ARDS	3	DEATH	DEATH	DEATH
OLIGURIC	3.1	97	ACIDOSIS	NA	NA	DEATH	NA	NA	ARDS	1	DEATH	DEATH	DEATH
NONOLIGURI	2.9	74	ACIDOSIS	NA	NA	DEATH	DEATH	NA	ARDS	1	1.1	NA	NA

NAME	AGE	SEX	EDUCATION	SOCIOECONOMIC	OCCUPATION	PLACE	NATIVE	SPECIES	Ist CARE	STINGS	REMOVED	REFERALLAG	BP	OUTPUT	PROTIENURIA	HEMATRIA	HB
YUGIN	21	M	1	1	ACCIDENTAL		NO	VESPEDIA	60	24	YES	6 HRS	HIGH	OLIGURIC	YES	YES	11.1
SIVASHANKAR	70	M	1	1	ACCIDENTAL		NO	VESPEDIA	40	30	YES	12 HRS	N	NON OLIGURIC	NO	NO	9.7
ravi	40	M	2	1	OCCUPATIONAL		NO	VESPEDIA	34	72	YES	7 HRS	HIGH	OLIGURIC	NO	YES	10.3
MOORTHI	38	M	2	1	OCCUPATIONAL		NO	VESPEDIA	20	42	YES	2HRS	N	OLIGURIC	NO	NO	11.8
MANAVALAN	85	M	1	1	ACCIDENTAL		NO	VESPEDIA	40	48	YES	11HRS	N	NON OLIGURIC	NO	YES	8.4
SETTU	25	M	1	1	ACCIDENTAL		TOPICAL	VESPEDIA	60	12	YES	24HRS	N	OLIGURIC	YES	NO	8.9
LAKSMI	27	F	3	2	ACCIDENTAL		NO	VESPEDIA	60	31	YES	4HRS	N	NON OLIGURIC	YES	NO	8.1
KANDASWAMY	36	F	2	2	OCCUPATIONAL		NO	ASPEDIA	90	NO	NO	2HRS	HYPOTENSION	OLIGURIC	NO	NO	11.1
DINESHKUMAR	22	M	1	1	OCCUPATIONAL		NO	VESPEDIA	60	45	YES	2HRS	N	NON OLIGURIC	NO	NO	12
CHINNASWAMY	35	M	1	2	OCCUPATIONAL		NO	VESPEDIA	60	43	YES	4HRS	N	NON OLIGURIC	NO	NO	11
DHANAPAL 73	73	M	1	2	ACCIDENTAL		TOPICAL	VESPEDIA	34	34	PARTIAL	7HRS	HYPOTENSION	OLIGURIC	NO	NO	11

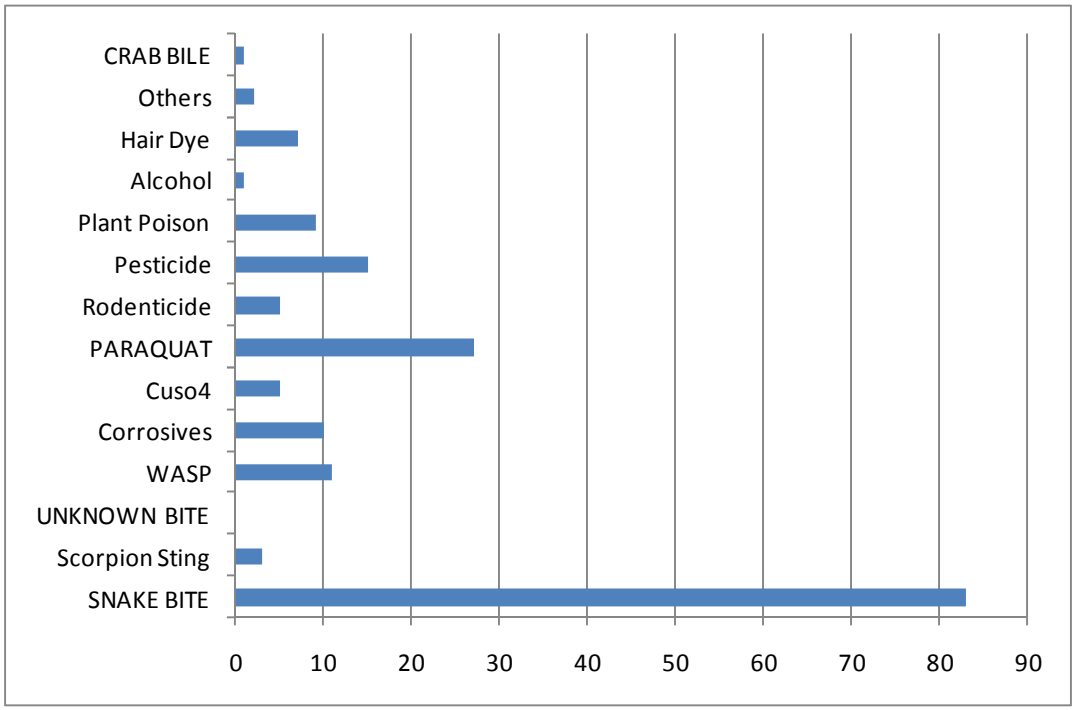
PLASMA HB	RETICONT	LDH	CPK	SR.BILRUBIN	OT	PT	PLATELET	PT	APTT	DIC	SR.K	ANAPHYLAXIS	ANGIOEDEMA	SHOCK	UTRCARIA	LIVER	COAGULOPATHY	RHABDOMYOLYSIS	RESPIRATORY	BL.UREA	II UREA
NO	NO	123	549	0.8	24	14	2.8	NO	NO	NO	3.7	NO	NO	NEUROGENIC	YES	NO	NO	YES	ARDS	112	145
NO	NO	128	36	0.9	22	12	2.6	NO	NO	NO	3.2	YES	NO	NO	YES	NO	NO	NO	NO	96	143
NO	NO	728	1328	0.8	28	32	2.4	NO	NO	NO	5.8	NO	NO	NEUROGENIC	YES	NO	NO	YES	NO	88	144
NO	NO	108	650	0.8	20	14	2	NO	NO	NO	4	NO	NO	NO	YES	NO	NO	YES	NO	94	132
NO	NO	340	983	1	28	22	2.8	NO	NO	NO	5.6	NO	NO	NO	YES	NO	NO	YES	NO	89	96
YES	ELEVATED	670	3220	2.1	34	23	0.8	PROLONGED	PROLONGED	YES	5.2	NO	NO	NO	YES	YES	YES	YES	NO	112	156
NO	NO	453	978	0.9	34	16	1.1	NO	NO	NO	3.9	NO	NO	NO	YES	NO	NO	YES	NO	78	92
NO	NO	344	588	0.8	23	12	1.3	NO	NO	NO	4.6	NO	NO	CARDIOGENIC	YES	NO	NO	YES	NO	110	154
NO	NO	440	877	1.3	23	22	1.1	NO	NO	NO	5.2	NO	NO	NO	NO	NO	NO	YES	NO	59	72
NO	NO	345	874	1.1	24	24	1.6	NO	NO	NO	4.9	NO	NO	NO	NO	NO	NO	YES	NO	55	66
YES	YES	567	3444	2.5	65	77	1.8	PROLONGED	PROLONGED	YES	5.4	YES	YES	YES	YES	YES	YES	YES	YES	145	188

FAD	HD/PD	DIA.TIME	SR.CRP	SR.CR	OUTPUT	INDICATION	TOTAL	DURATION	BIOPSY	STERIODS	RESPONSE	IMPR.OUTPUT	BASELINE	DEATH	CAUSE OF DEATH	TIME OF DEATH	VENTILATOR	DIS.CR	FOLLOW UP EGFR	LATEST	special	steriod	response
NO	HD	1ST	13	13	OLIGURIC	RHABDO	5	11	NOTDONE	NO	NA	6	13	NA	NA	NA	NA	1.1	1.1	N	NO	NO	NO
YES	HD	IST	7.6	7.6	OLIGURIC	ACIDOSIS	10	22	AIN	YES	GOOD	20	24	NA	NA	NA	NA	1.7	0.6	N	NO	yes	good
YES	HD	IST	8.7	8.7	OLIGURIC	RHABDO	14	20	NOTDONE	NO	NA	21	24	NA	NA	NA	NA	1.4	0.9	N	myoglobu	NO	NO
YES	HD	IST	8.4	8.4	OLIGURIC	RHABDO	5	9	ATN	NO	NA	11	14	NA	NA	NA	NA	1.1	0.9	N	NO	NO	NO
YES	HD	IST	6.9	6.9	ANURIC	RHABDO	18	33	ATN/AIN	YES	GOOD	23	27	NA	NA	NA	NA	1.1	1.1	N	NO	NO	NO
YES	HD	2ND	9.6	9.6	OLIGURIC	ACIDOSIS	NOT RECOVERED	NOTRECOVERED	ATN/AIN	YES	GOOD	CKD	ONMHD	YES	SEPSIS	4 M0ONTHS	NA	4.2	1.2	lost follow up	NO	NO	defaulter
YES	HD	IST	5.4	5.4	OLIGURIC	RHABDO	11	23	ATN/AIN	NO	GOOD	18	24	NA	NA	NA	NA	2.1	1.1	N	NO	NO	good
YES	HD	IST	6.3	6.3	ANURIC	ANURIA	6	10	NOTDONE	NO	NA	8	11	NA	NA	NA	NA	1.6	1.1	N	NO	NO	NO
YES	NO	NO	1.7	1.9	NONOLIGURIC	RHABDO	NO	NO	NOTDONE	NO	NA	NA	NA	NA	NA	NA	NA	1.1	0.9	N	NO	NO	NO
YES	NO	NO	1.9	1.9	NONOLIGURIC	RHABDO	NO	NO	NOTDONE	NO	NA	NA	NA	NA	NA	NA	NA	1.2	0.9	N	NO	NO	NO
NO	HD	1ST	6.7	8.3	ANURIC	RHABDO	2	2	NOTDONE	NO	NA	NA	NA	DEATH	DIC	2ND	YES	DEATH	1	n	myoglobu	NO	

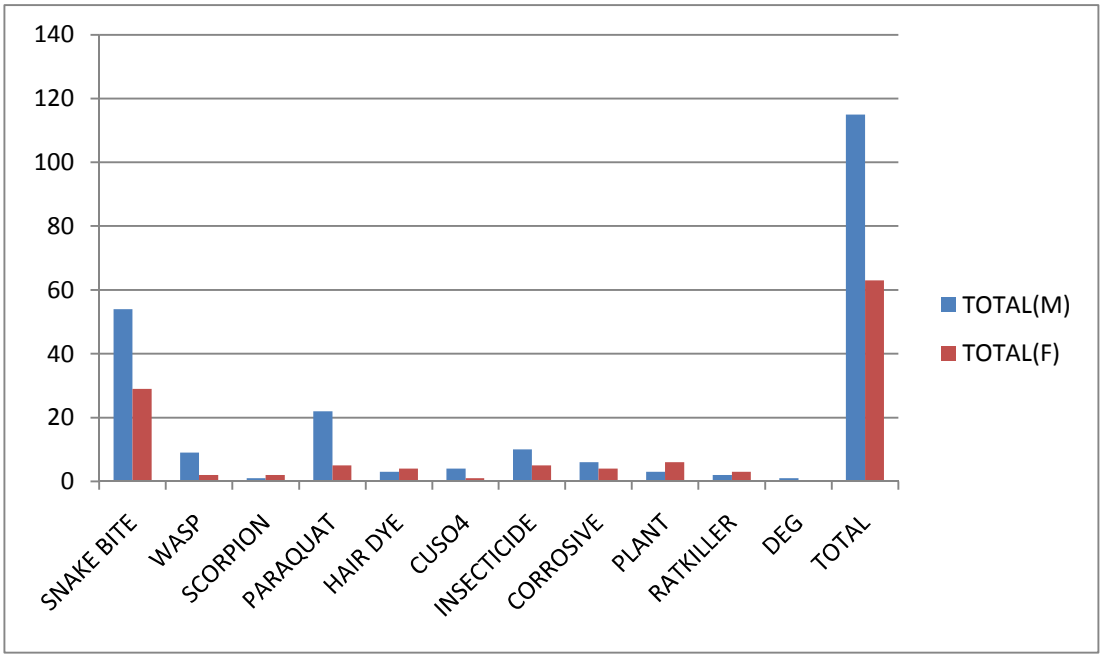
TOTAL POISONING CASES



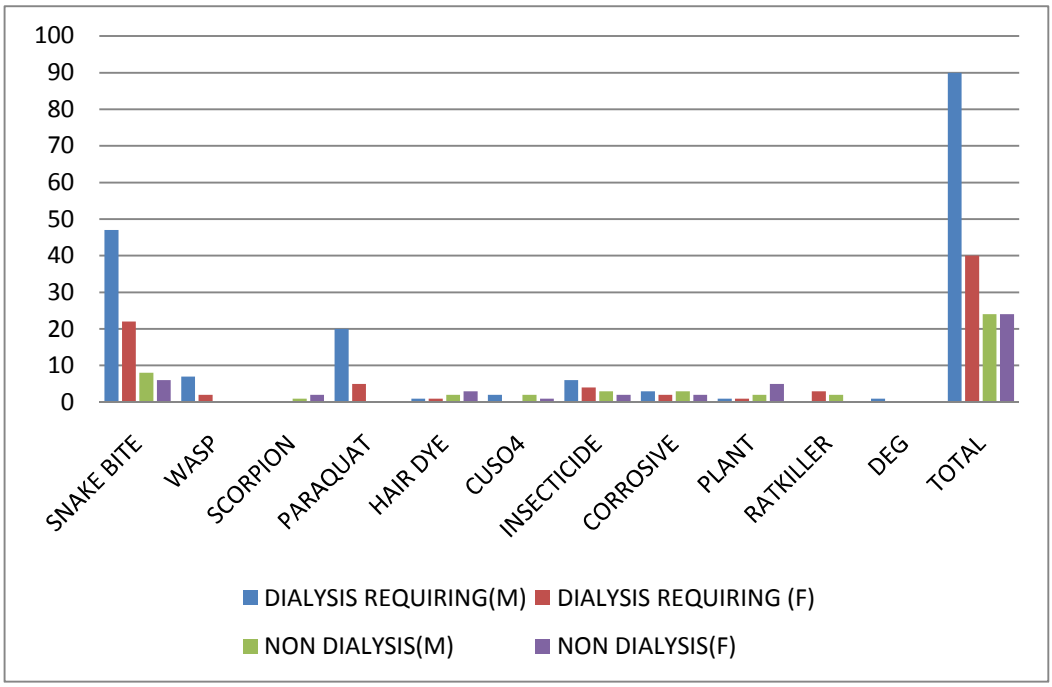
TOTAL AKI

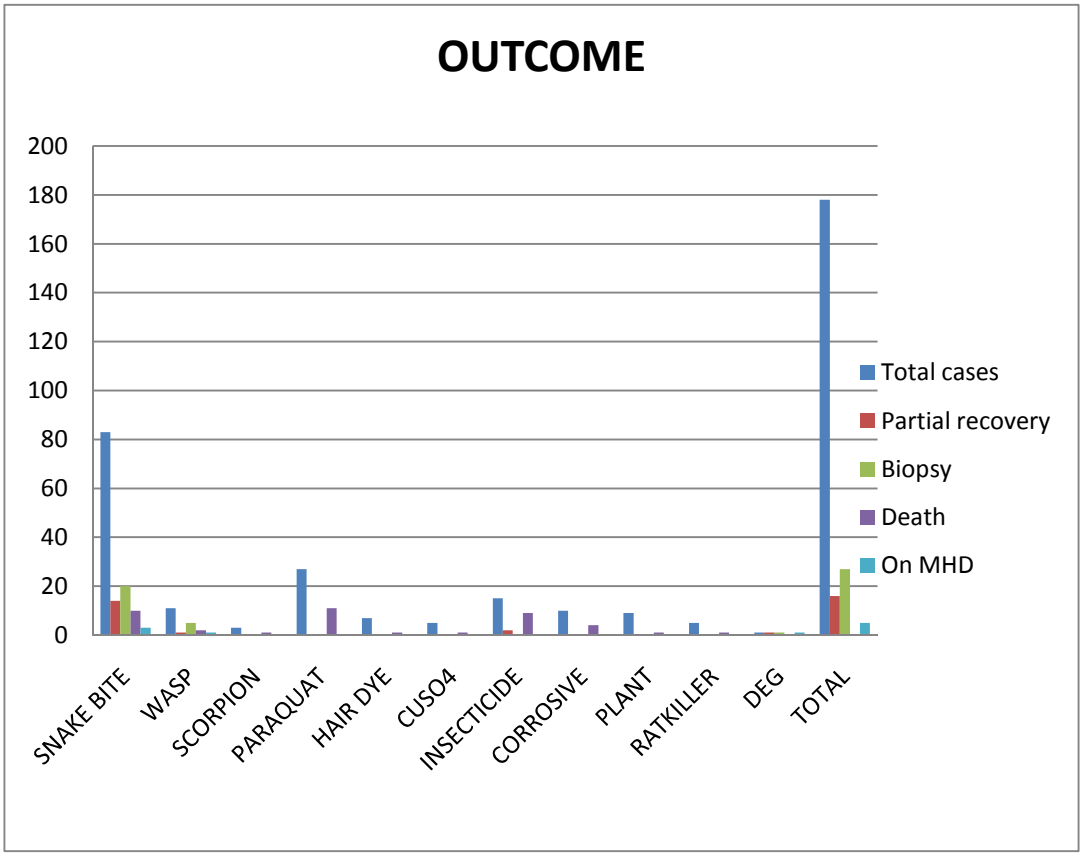


SEX DISTRIBUTION

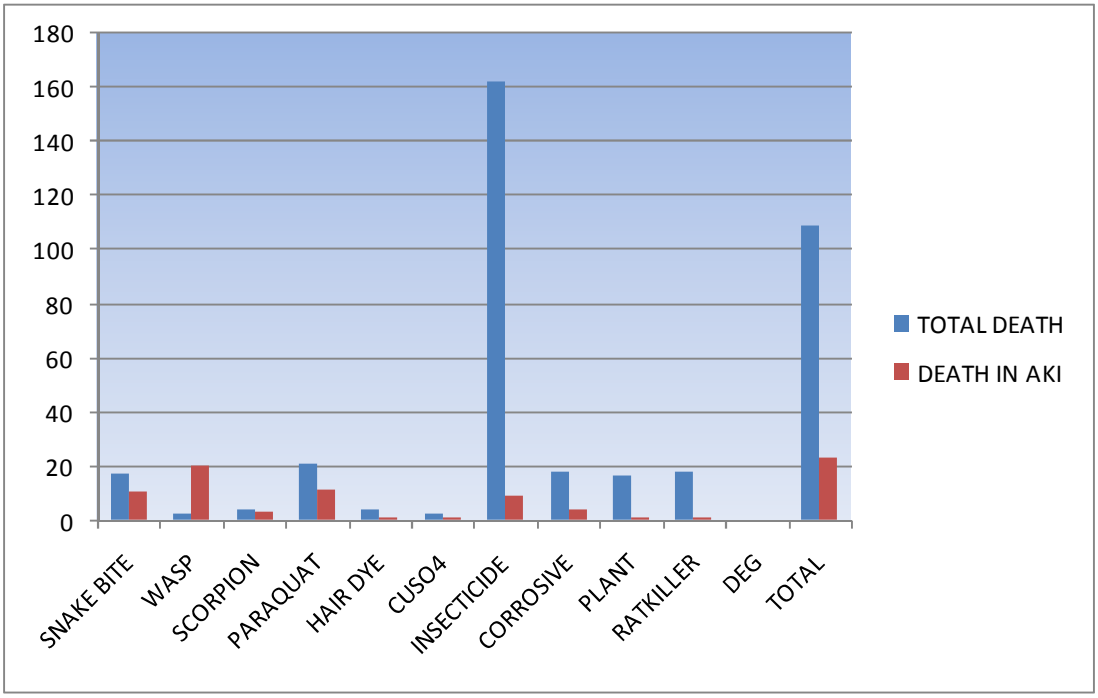


DIALYSIS REQUIREMENT

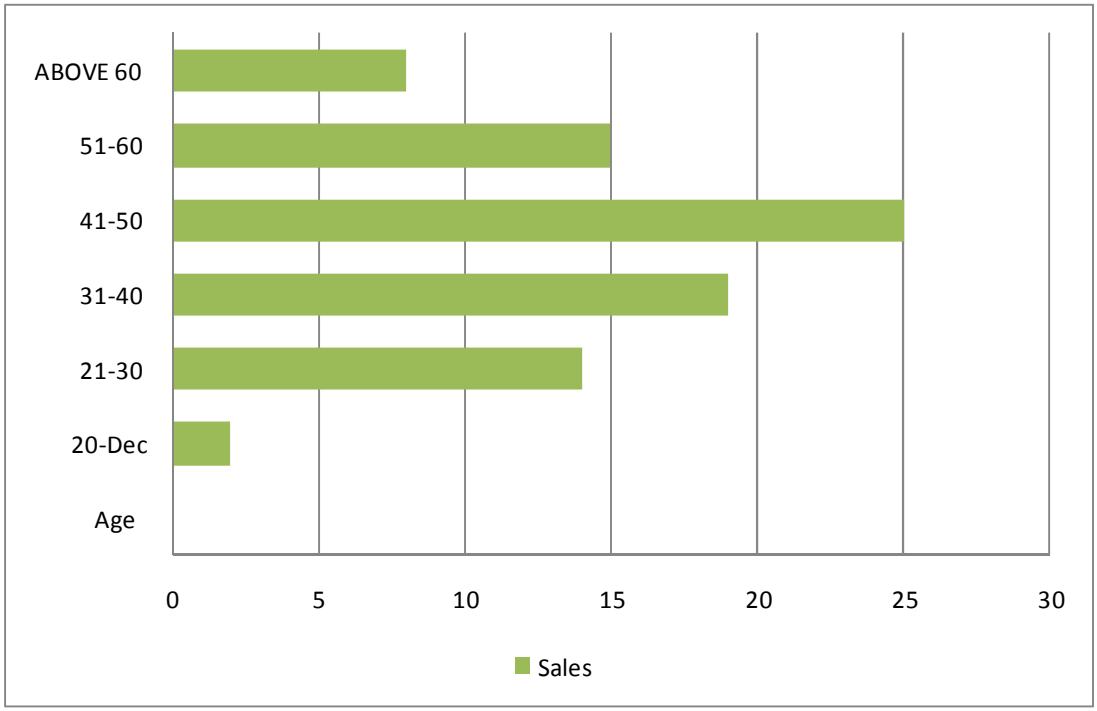



OUTCOME
200
180
160
140
120
100
80
60
40
20
0
Total cases
Partial recovery
Biopsy
Death
On MHD
SNAKE BITE
WASP
SCORPION
PARAQUAT
HAIR DYE
CUSO4
INSECTICIDE
CORROSIVE
PLANT
RATKILLER
DEG
TOTAL

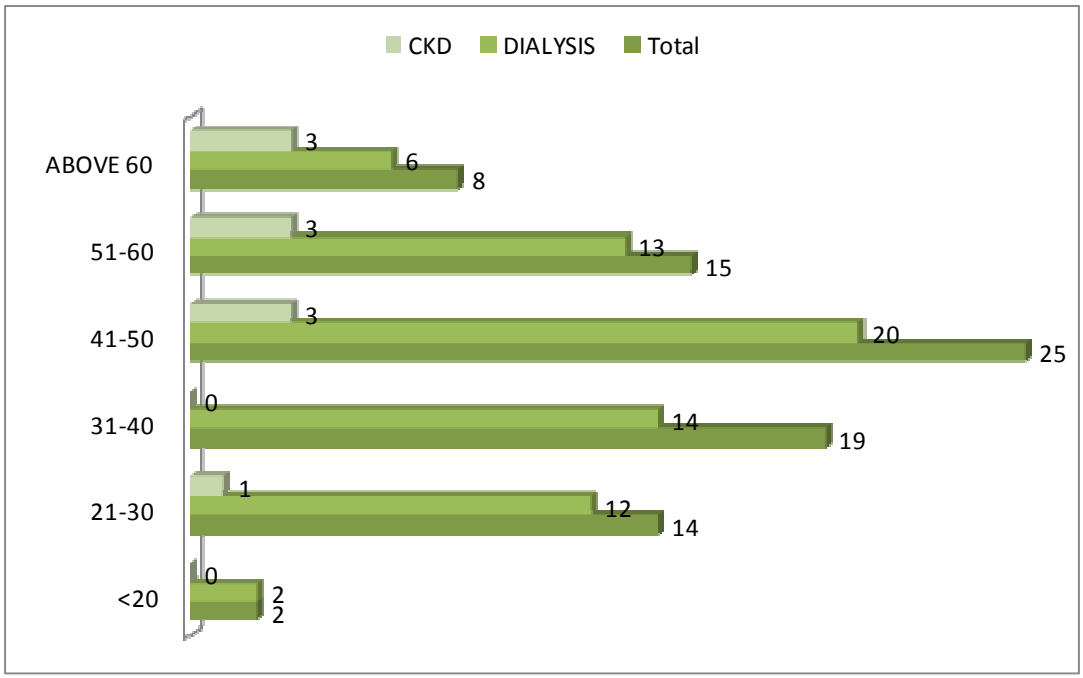
MORTALITY


180
160
140
120
100
80
60
40
20
0
TOTAL DEATH
DEATH IN AKI
SNAKE BITE
WASP
SCORPION
PARAQUAT
HAIR DYE
CUSO4
INSECTICIDE
CORROSIVE
PLANT
RATKILLER
DEG
TOTAL

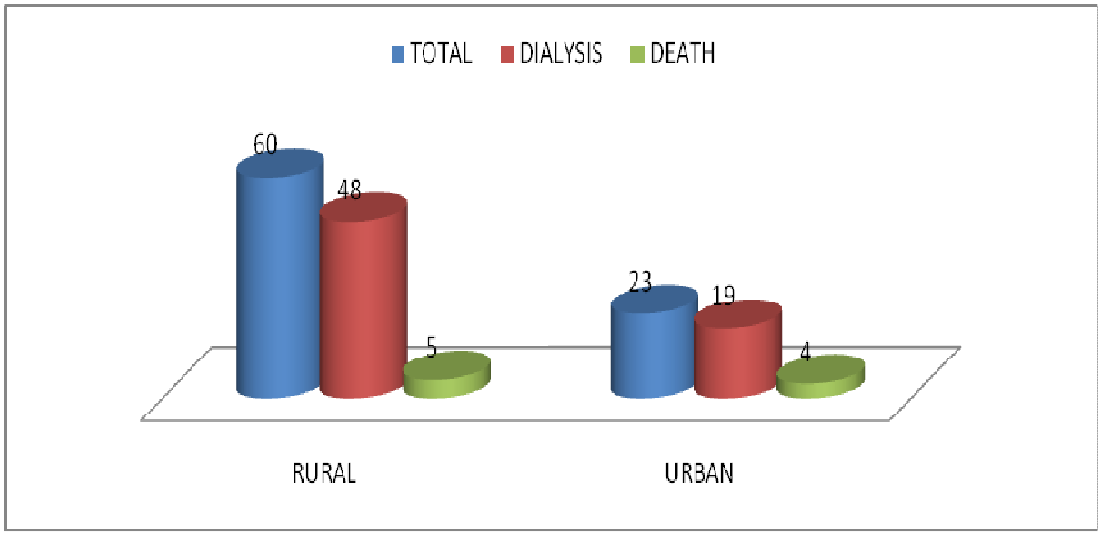
SNAKE BITE AGE DISTRIBUTION

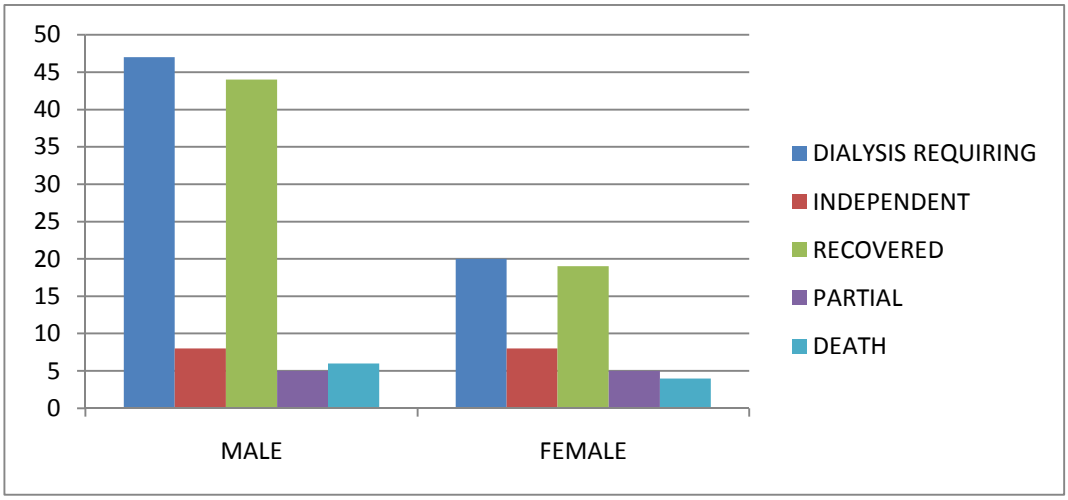


AGE WISE OUTCOME IN SNAKE BITE

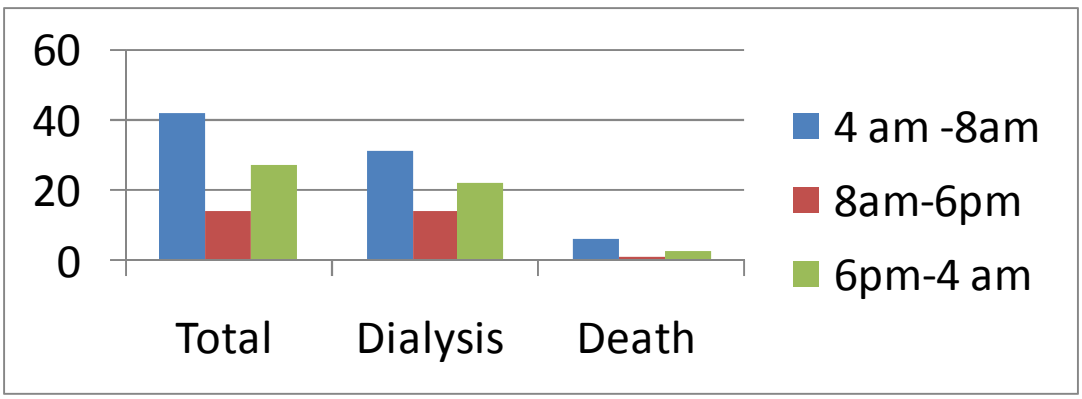


SNAKE BITE



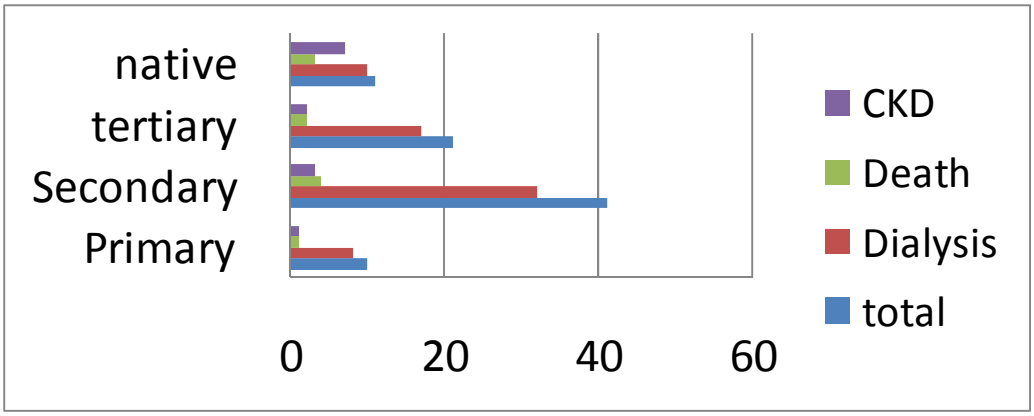


TIMING OF BITE AND OUTCOME

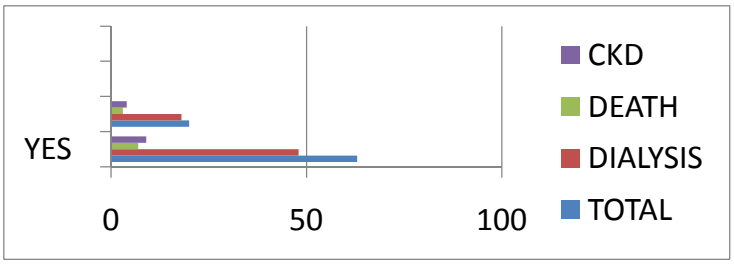


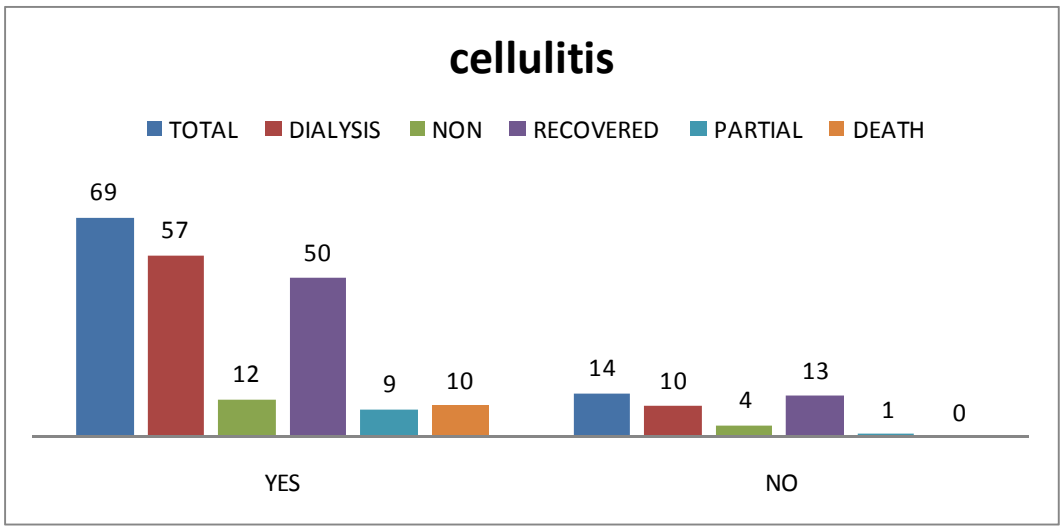
SNAKE BITE

LEVEL OF CARE AND OUTCOME:

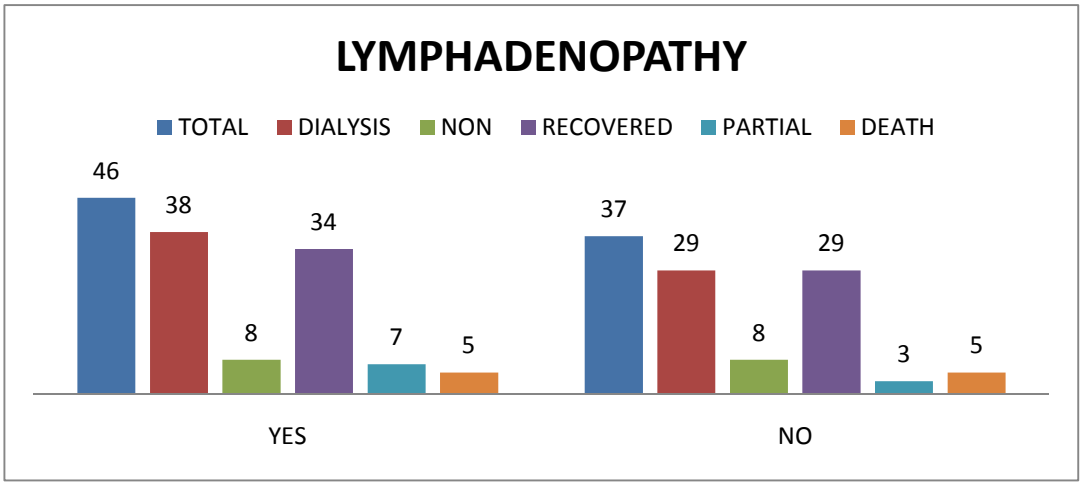


NATIVE MEDICATION AND OUTCOME

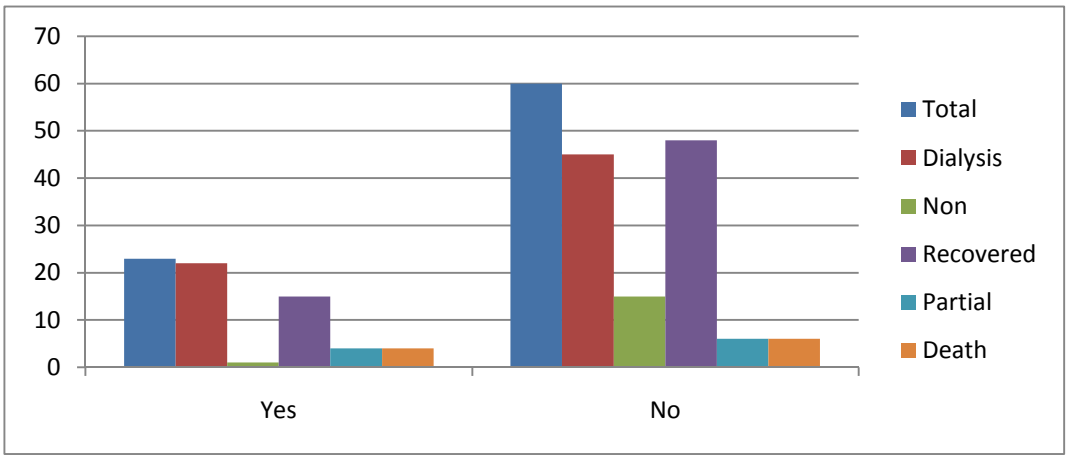




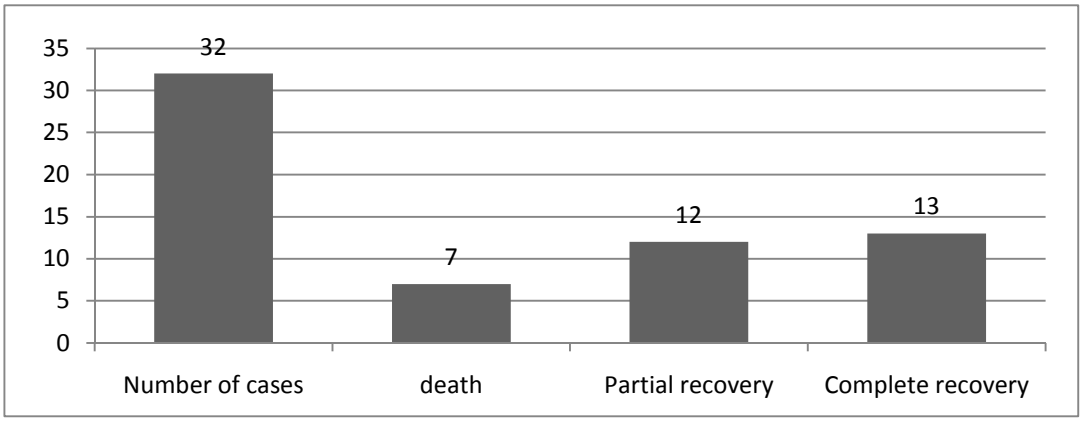
SNAKE BITE



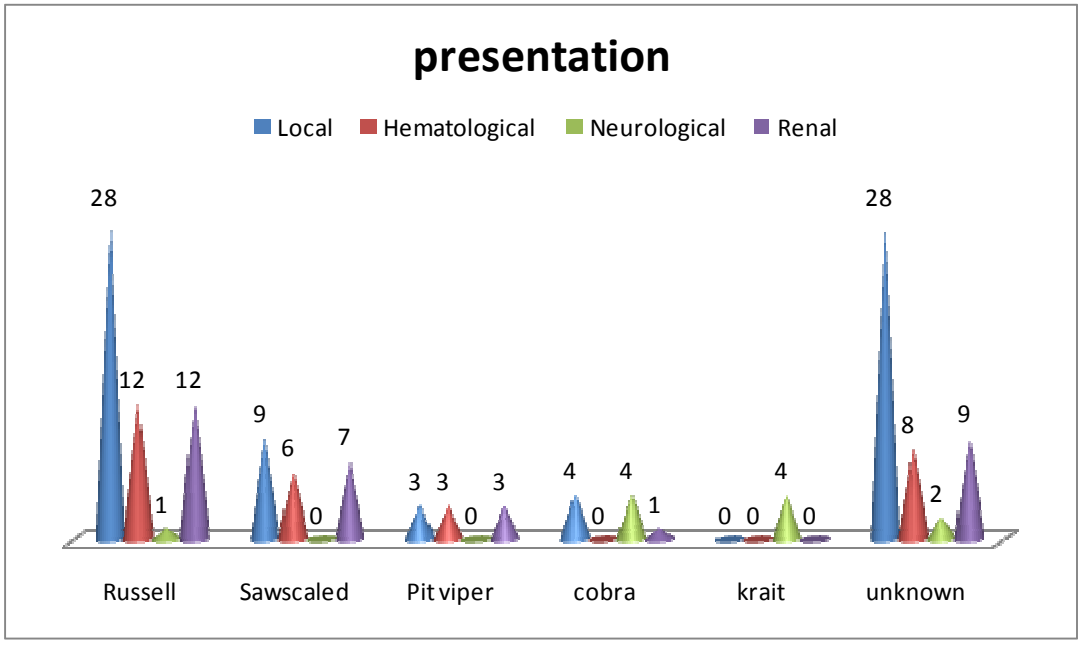
FANG MARKS AND OUTCOME



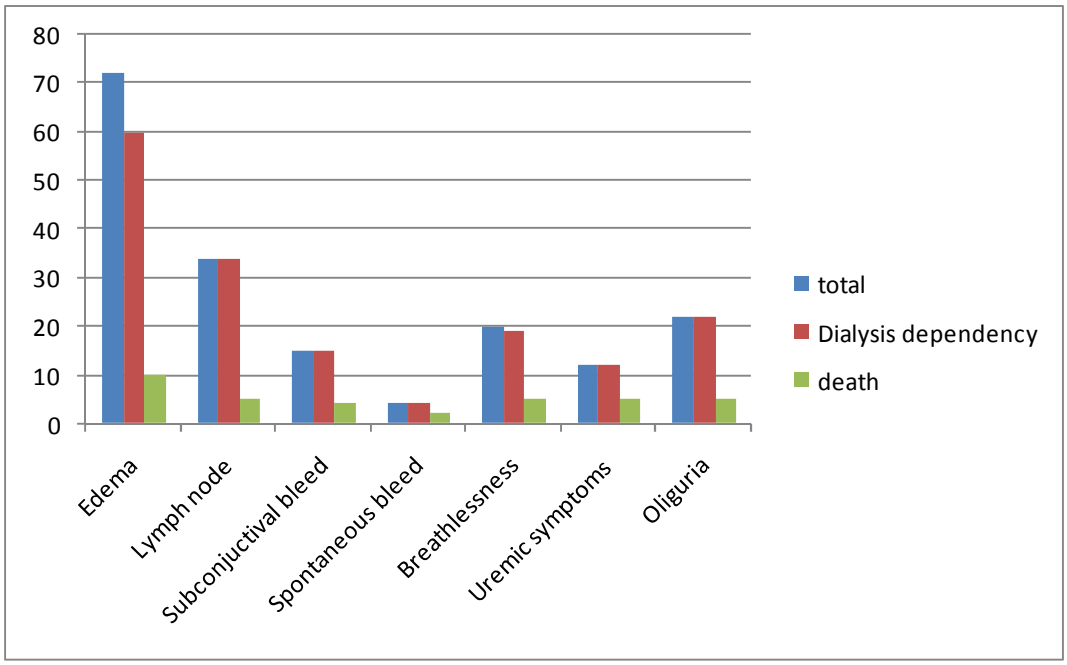
RENAL FAILURE AT PRESENTATION AND OUTCOME:



SNAKE BITE

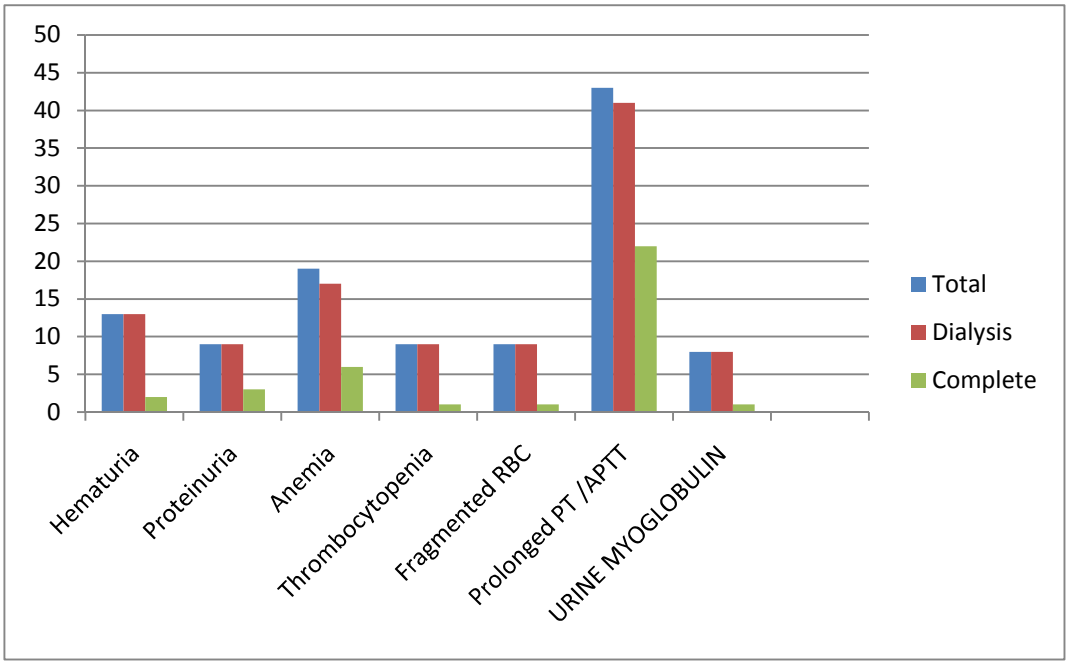


SYMPTOM ANALYSIS

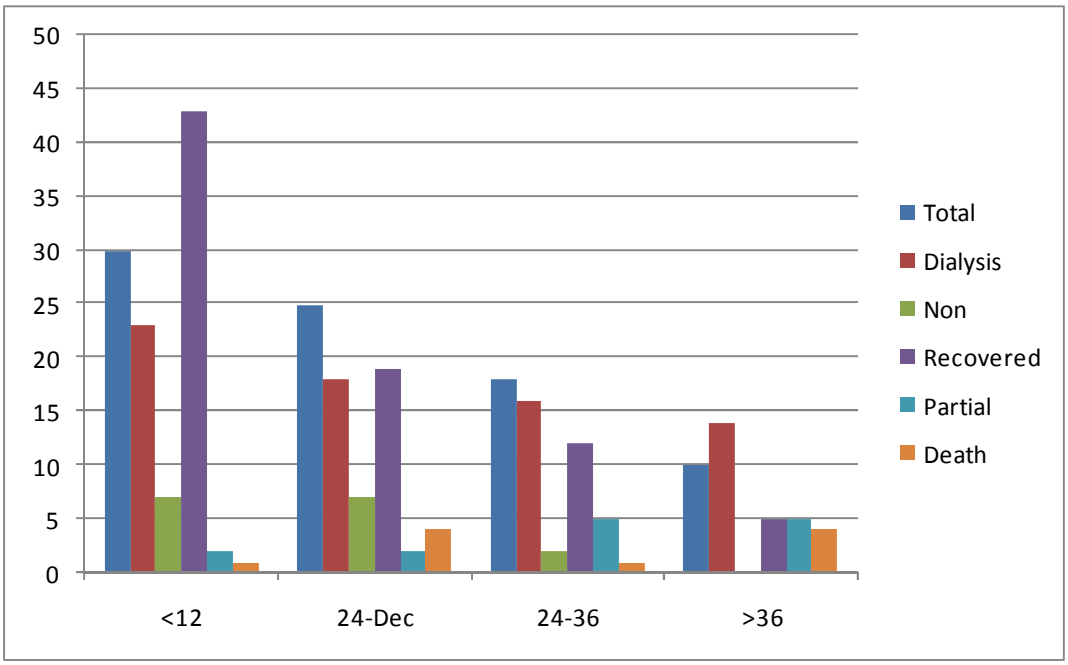


SNAKE BITE

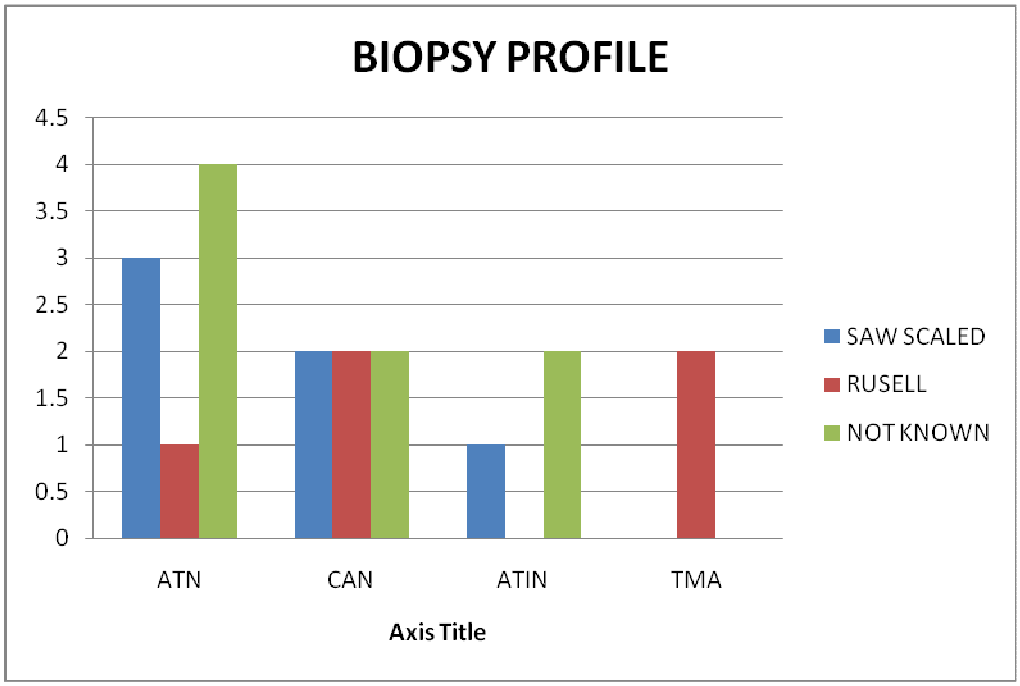
LABAROTARY PARAMETERS

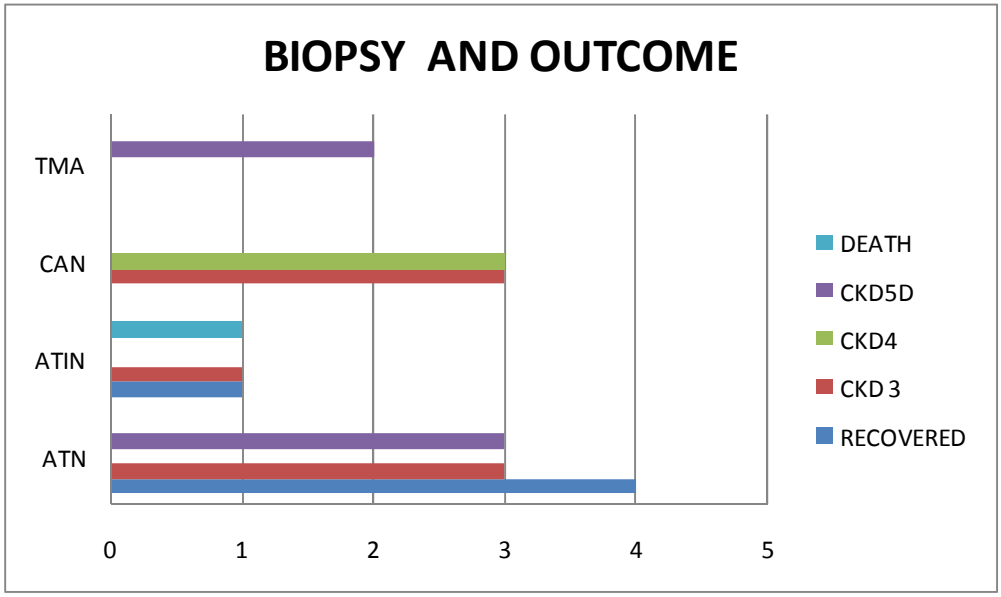


TIME OF NORMALIZATION OF WBCT

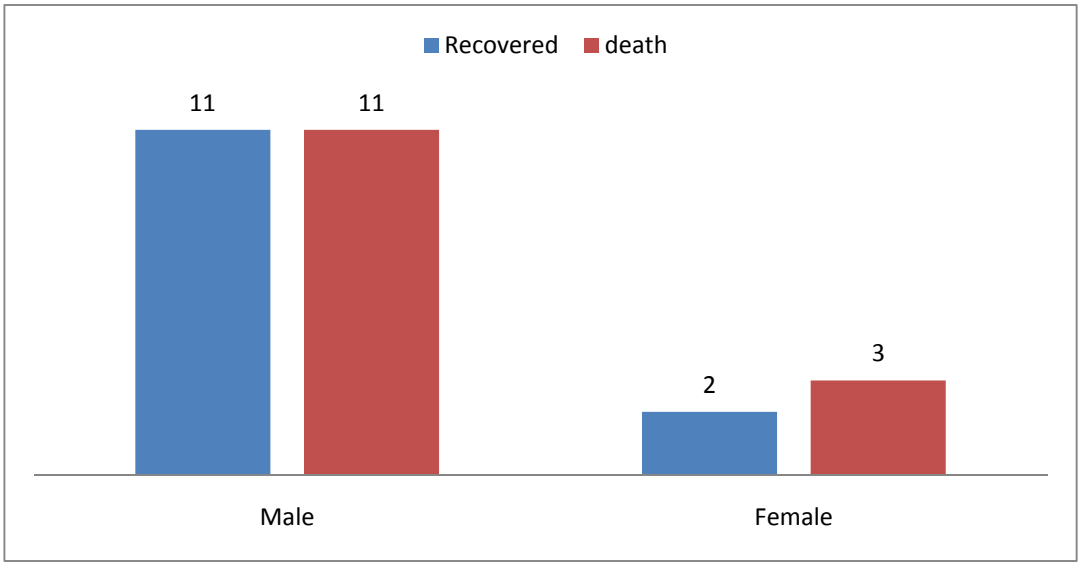


SNAKE BITE

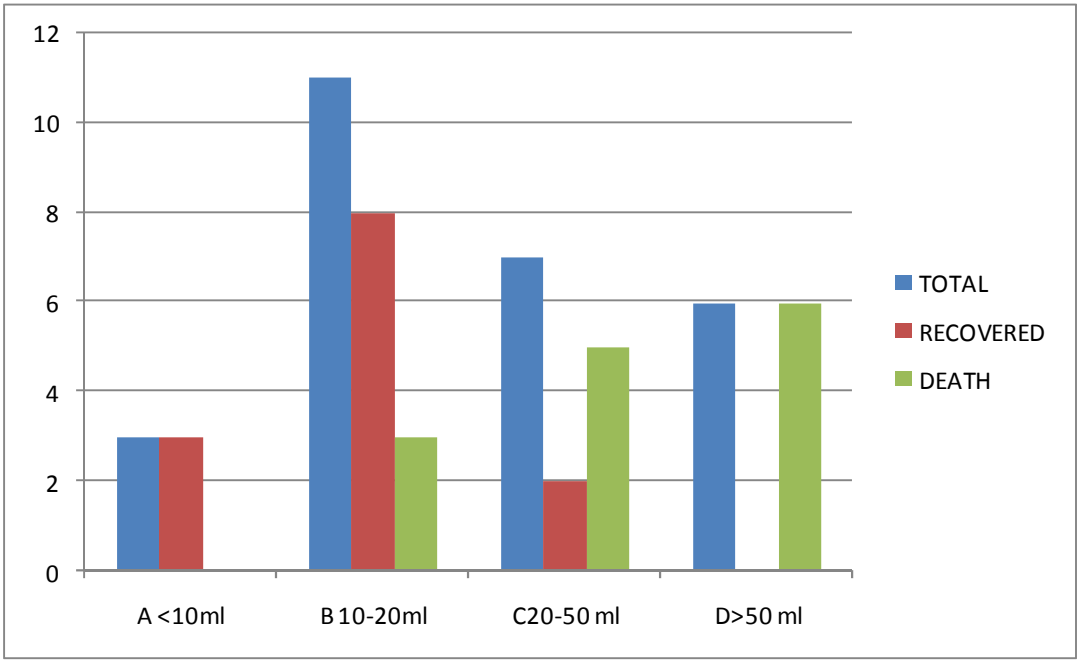

BIOPSY PROFILE
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
ATN
CAN
ATIN
TMA
Axis Title
SAW SCALED
RUSELL
NOT KNOWN


BIOPSY AND OUTCOME
TMA
CAN
ATIN
ATN
0
1
2
3
4
5
DEATH
CKD5D
CKD4
CKD 3
RECOVERED

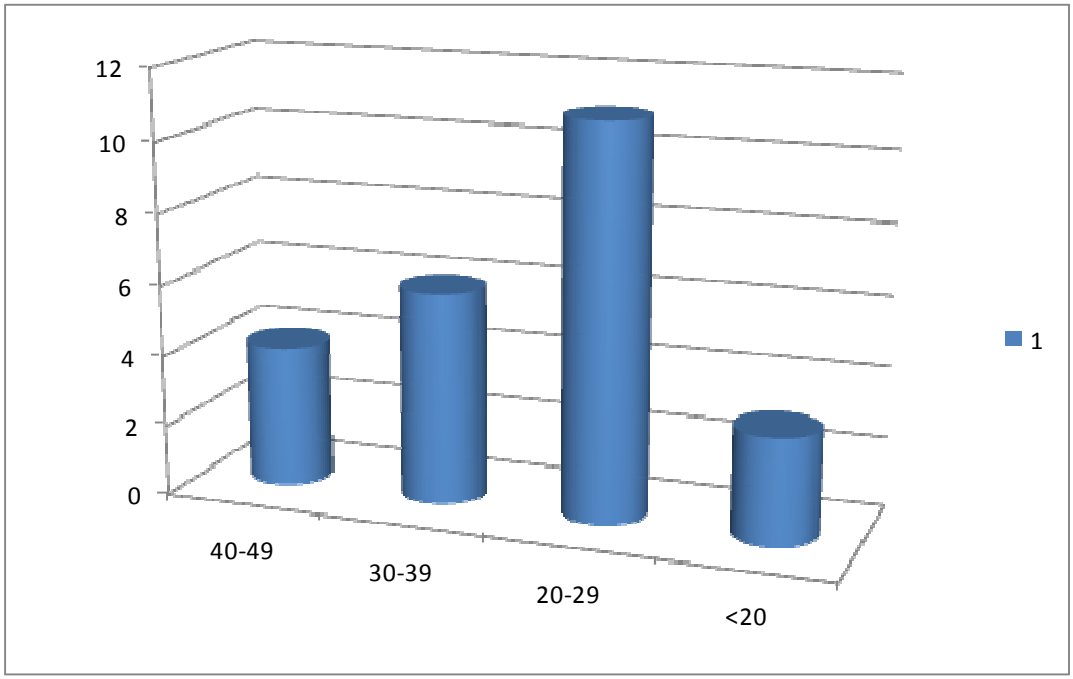
PARAQUAT



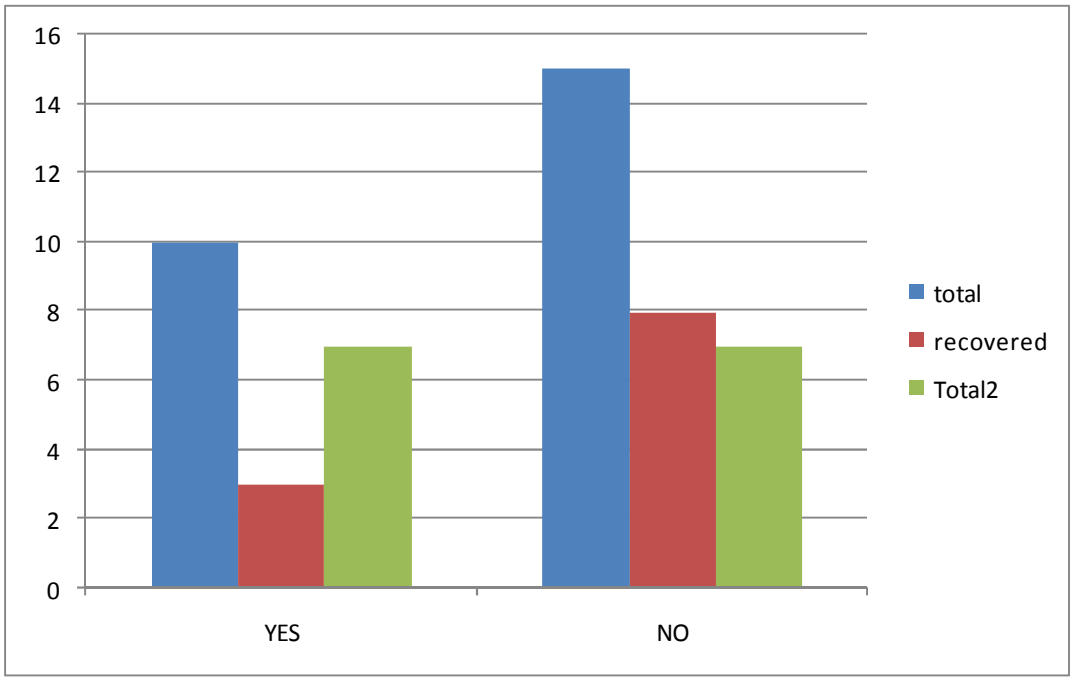
Amount of poison and Outcome



AGE WISE DISTRIBUTION

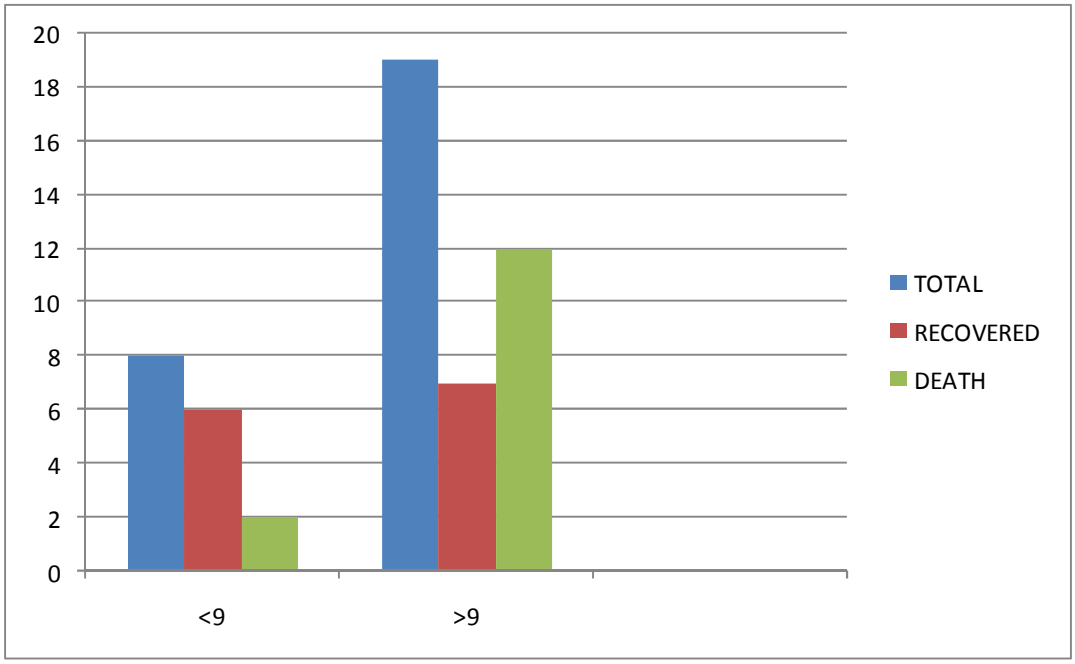


Preemptive dialysis and Outcome

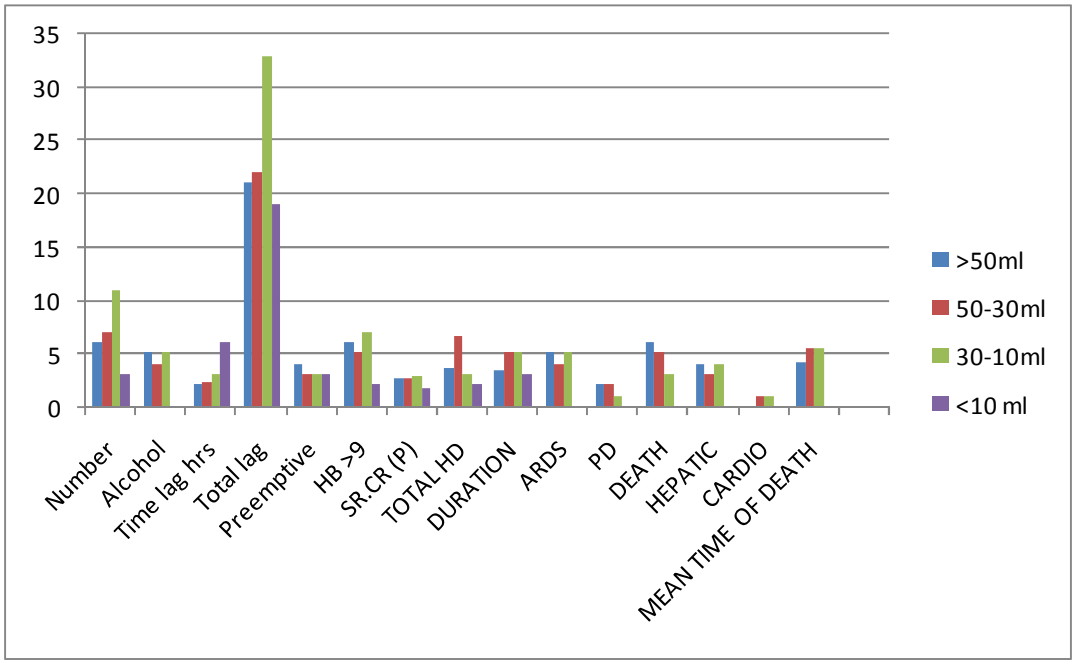


PARAQUAT

Baseline hemogobulin vs Outcome



AMOUNT AND OUTCOME



INSECTICIDE